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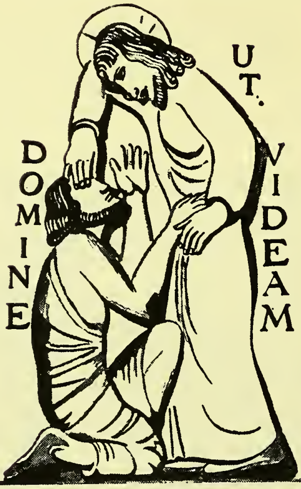

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AN INQUIRY
INTO THE PHYSIOLOGY AND PATHOLOGY OF THE INTRA-OCULAR PRESSURE

BY

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Gold Medallist, Eleventh International Ophthalmological Congress, Naples, 1909

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1910

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TO

LEONARD HILL, M.B., F.R.S.

LECTURER ON PHYSIOLOGY, LONDON HOSPITAL MEDICAL SCHOOL

IN GRATEFUL APPRECIATION

OF HIS DISCOVERIES OF THE PRINCIPLES

GOVERNING THE PHYSIOLOGY AND PATHOLOGY

OF THE

CEREBRAL CIRCULATION

PREFACE

The aim of the present work is to bring forward sufficient cumulative evidence to establish the circulatory nature of the Intra-ocular pressure, and thus to correlate the conditions in the eye with those already proved by Leonard Hill as existing in the brain. The task, at the outset a difficult one, is made still more so by the fact that such a conception of the intra-ocular pressure is totally opposed to current views, thereby making the field to be covered so extensive that there is considerable danger of obscuring, in a wearisome infinity of details, the underlying and all-important physiological principles.

These physiological principles amply confirm the view of the pathogenesis of Primary Glaucoma which I first put forward in a preliminary communication read before the Ophthalmological Section of the British Medical Association at Exeter in 1907.

To the authorities of the London Hospital Medical School I am indebted for generous permission to conduct my experimental work in their premises, where I had the kind and invaluable co-operation of Dr. Leonard Hill. For much valuable pathological material I have to thank numerous friends, particularly Professor Fuchs of Vienna, Mr. Richardson Cross, Mr. Skeffington Cresswell, and Dr. Wood of Hereford.

To Dr. Goldsmith of Bedford I am under much obligation for the skilful execution of certain of the drawings, as well as for assistance in the revision of proofs. Finally, I have to acknowledge the courtesy of the editors of *The Ophthalmic Review* and of *The Ophthalmoscope* for the use of the blocks of illustrations which have appeared in those journals.

15, The Ropewalk,
Nottingham,
September, 1910.

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GLAUCOMA

PART I

ANATOMY

The Eyeball as a Closed Sphere.

The general distribution of the intra-ocular contents, apart from their purely optical purpose, will be much easier to grasp if certain physiological conditions, relative to the eyeball as a closed sphere, are at the outset clearly recognized and prominently kept in view.

To this end a preliminary survey of the problems connected with the nature of the intra-ocular pressure and the maintenance of the form of the eye will materially assist.

According to current conceptions,* the closed corneoscleral envelope represents a sphere the maintenance of whose form depends on the pressure exerted on the fibrous capsule by its contents. The normal intra-ocular pressure, according to this hypothesis, depends on the quantity of the fluids within the globe being kept constant—in other words, it is purely a question of volume. Variations in the volume of the intra-ocular contents give rise to alterations in intra-ocular pressure, and therefore the globe of the eye has come to be regarded as an elastic capsule, whose capacity, form, and internal pressure, depend on the balance struck between a constant inflow, or formation of aqueous, and a proportionate outflow or resorption.

---

* See Parsons' "Pathology of the Eye," chap. xx.

The numerous objections to this theory need not now detain us ; suffice it to state that the application to the eye of the magnificent experimental work of Leonard Hill* on the nature of the intra-cranial pressure completely disproves this volumetric conception of the intra-ocular pressure. Leonard Hill has shown that in the closed and rigid cranium the intra-cranial pressure is the same as the cerebral venous pressure—in other words, it is that pressure which remains after the force of the heart has been expended in driving the blood through the cranial arterioles. It is therefore an ever-varying quantity, depending directly on the general circulation.

In the eyeball the conditions which give rise to the intra-ocular pressure are exactly similar to those which exist in the cranium. The identity of the pressure within these two closed spheres is conclusively demonstrated by the simultaneous readings given by manometers connected respectively with a brain pressure gauge, and with a hollow needle inserted into the Anterior Chamber. The hydrostatic pressures within the enclosing coverings of the eye and brain rise and fall together under all varying physiological and experimental conditions, thus definitely and absolutely proving that the intra-ocular pressure is not determined by intra-ocular volume, but, like the intra-cranial, it too is vascular in origin and nature, and stands under all physiological conditions likewise at intra-venous pressure.

The intra-ocular and intra-cranial pressures being identical, their coverings must have exactly the same physiological properties. The closed corneo-scleral envelope behaves, in fact, not as an elastic capsule, but as a rigid and unyielding case, which, being composed of fibrous tissue, is not as firm as the shell of bone forming the skull ; yet its cubic capacity, like that of the cranium, is strictly limited. The maintenance of the form of the normal and living eye thus depends, not on the pressure exerted from within, but on the rigidity of its outer covering. Like the cranium, the unyielding connective-tissue capsule of the eyeball has a fixed cubic capacity, and its contents being incompressible, there can be no question of variations in the total intra-ocular volume being able to induce changes of intra-ocular pressure. The intra-ocular volume is fixed, but the pressure of the fixed volume varies, depending on circulatory conditions. This aspect of the nature of the intra-ocular pressure is thus totally at variance with recognized physiological teaching, and therefore it is necessary to grasp the fundamental difference between the two points of view, as all the results of the researches which follow are considered in their relation to the vascular conception of the intra-ocular pressure.

---

* "The Physiology and Pathology of the Cerebral Circulation," 1896.

The point that I would here emphasize is that the normal intra-ocular pressure is the same as the intra-cranial pressure, and that like this latter it stands and varies with the venous pressure within its enclosing covering, which pressure is in its turn maintained and influenced by the general circulation (*vide infra*).

The identity of the intra-ocular and intra-cranial pressures permits the nervous elements subserving the visual function remaining throughout their whole course under the same hydrostatic pressure (Fig. 1). The *Lamina Cribrosa* can therefore well afford to be the weakest spot in the corneo-scleral envelope, as under all physiological conditions the same hydrostatic pressure exists on the one side of it as on the other. The physiological parity of the two pressures is at the same time associated with an exact anatomical analogy in the disposition of the nerve elements in relation to their unyielding coverings. The nerve elements of eye and brain are respectively bounded by the water-beds

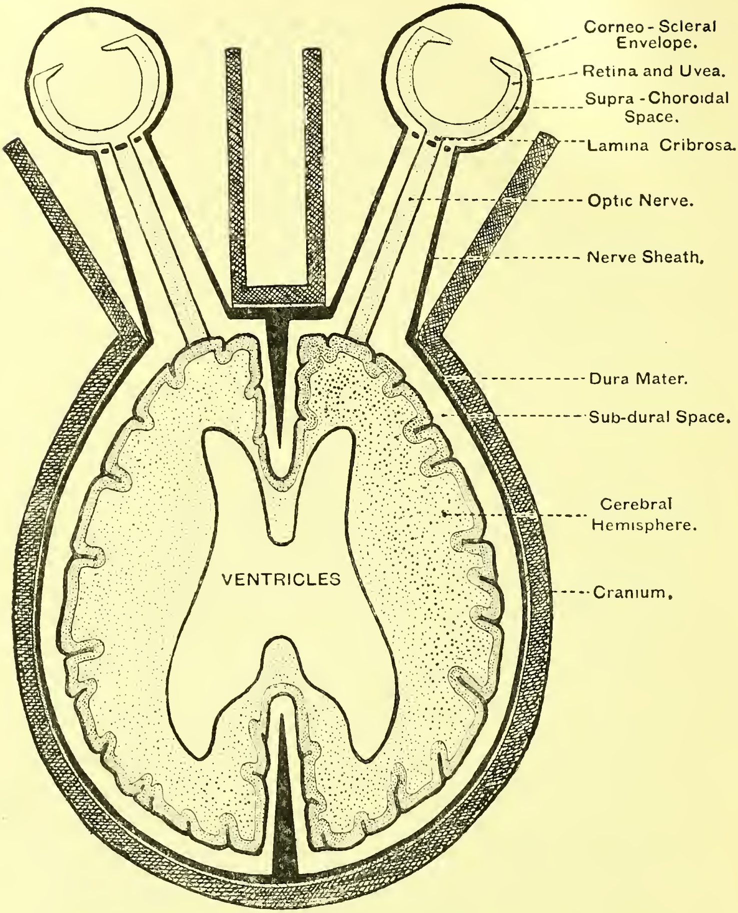

Fig. 1.—Analogous Disposition of the Intra-Ocular and Intra-Cranial Contents.

The nerve elements of the Eye (the retina) and of the Brain (the cerebral hemispheres) are arranged as hollow shells, containing respectively the Vitreous chamber and lateral Ventricles, the whole being enclosed by the unyielding outer coverings of the fibrous corneo-sclera and the bony cranium. The hydrostatic pressure within these two envelopes stands under all physiological conditions at the same intra-venous level; hence the structural weakness of the *Lamina Cribrosa*, as it is not required to withstand any pressure. To prevent injurious contact between nerve elements and their outer coverings are interposed the water-beds of the Supra-Choroidal and Sub-dural spaces, as well as the vascular beds of the Uvea and Pia Mater. The counterpart of the fluid reservoirs of the Aqueous Chambers of the Eye is represented in the Cranium by the basal *Cisternæ Sub-arachnoidales.*

of the Supra-choroidal and Sub-dural Spaces, which so surround and enclose the tissues that physiological variations in pressure can be instantly transmitted equally in all directions without any disturbing effect.

The fluid in these lymph spaces, while small in quantity, is of great physiological importance, for, being interposed between the nerve elements and their encircling rigid coverings, it prevents direct contact between the two. The intra-ocular and intra-cranial contents thus lie within rigid and closed walls, but are not pressed up against them.

When the central fact is kept in mind that the intra-ocular pressure is vascular in origin and nature, and that it stands and varies with the intra-cranial pressure, then the *raison d'être* of the anatomical disposition of the structures concerned with the formation and resorption of the intra-ocular fluids is much easier to comprehend.

The modifications from the recognized textbook teaching which I describe in the anatomical distribution of the intra-ocular structures would by themselves be of little value but for the fact that they are directly subservient to the vascular nature of the intra-ocular pressure; hence the importance of the early appreciation of this fundamental physiological conception.

Construction of the Angle of the Anterior Chamber.

In studying microscopic sections of the angle of the Anterior Chamber, there are two important points that require careful attention if a correct idea of the arrangement and relative position of its structures is to be obtained. The first point to be noted is the alteration in its topography induced by the artificial detachment of the Ciliary Body consequent to fixation in Müller's fluid, while the second and equally important point to be remembered is that the structures forming and bounding the Angle are disposed at the circumference of a circle, and therefore a correct perspective of their connections and interrelationships can only be obtained in sections which run truly radially.

Structures sectioned obliquely will appear under the microscope either fore-shortened or fore-lengthened, on which account random sections will tend to give an altogether wrong impression of the distribution and course of the individual fibres concerned. To overcome the difficulty produced by unavoidable obliquity, sections should be mounted and studied in series, as in this way not only can the imperfections in one section be noted, and corrected by the next, but the series when put together will reconstruct fibre for fibre the part sectioned as it actually existed, if not in the living eye, at any rate in the hardened and fixed globe.

The hardening of an eye in such a slow fixing medium as Müller's Fluid is followed, as is well known, by an artificial detachment of the Ciliary Body, but no allowance has ever been made for the effect such a detachment has in altering the form and appearance of the Angle of the Anterior Chamber.

It is only when the Angle in such globes is compared with the appearance and shape of the Angle in eyes in which displacement of the Ciliary Body does not occur, as after fixation in a rapid reagent such as Formalin, that the full effect of the alterations produced by detachment can be appreciated. Figs. 2 and 3 illustrate the great difference in the shape of the Angle of the Anterior Chamber in eyes hardened respectively in Formalin and Müller's Fluid.

The exclusive examination heretofore of globes fixed in such slow hardening reagents as Müller's Fluid is responsible for the classic presentation of the Filtration angle as an annular sinus; and as considerable stress is necessarily laid on this sinus formation in Priestley Smith's theory of

Glaucoma, it will be necessary to consider carefully how this appearance is produced.

In an eye fixed in Formalin, the Ciliary Body remains in position, and therefore *a priori* one would expect that the relative position of the structures about the Angle should be correct. This is undoubtedly the case. In such an eye (Fig. 2), the Angle presents itself as a true Angle (*a*, *b*, *d*), formed by the attachment (at *b*) of the Iris Root (*d*) to the Cribriform (so-called Pectinate) Ligament (*a*). A line connecting the posterior end of Schlemm's Canal with the most anterior and internal of the circular bundles of the Ciliary Muscle falls altogether outside the Angle of the Anterior Chamber. Such a base line in a

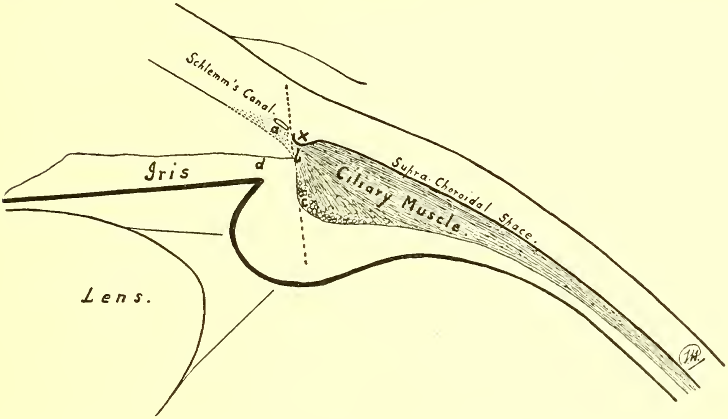

Fig. 2.—The Angle of the Anterior Chamber in a Formalin-fixed Globe.

The circumference of the Anterior Chamber forms a true angle (*a*, *b*, *d*), formed by the attachment (at *b*) of the Iris Root (*d*) to the Cribriform Ligament (*a*). A line joining the posterior extremity of Schlemm's Canal with the anterior and most internal of the circular bundles (*c*) of the Ciliary Muscle falls altogether outside the Angle.

The muscular bundles of the Ciliary Muscle form, with their intervening interstitial tissue, a compact structure lying in close apposition to the sclera, so that the Supra-Choroidal Space is seen, as it actually exists, as a mere potential space. The Scleral Ring (×) lies behind Schlemm's Canal.

globe where detachment of the Ciliary Body has followed fixation in Müller's Fluid will, however, fall within, instead of without, the Angle (Fig. 3). The greater the detachment of the Ciliary Body, the more markedly will this base line traverse the sinus-shaped region of the "filtration area," proving thereby the influence which this artificial condition has in altering the interrelationship of the structures about the angle.

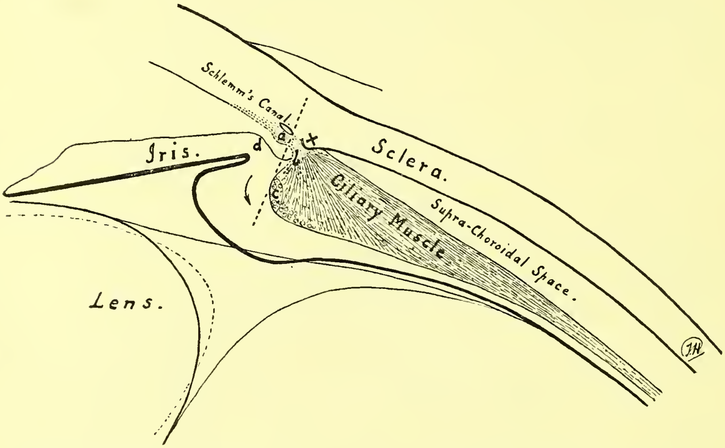

Fig. 3.—The Angle of the Anterior Chamber after Fixation in Müller's Fluid.

The Angle appears as a sinus (*a*, *b*, *d*) bounded on the outer side by the slackened fibres of the Cribriform Ligament (*a*), and on the inner by a bending (*d*) of the Iris Root, both conditions being the result of detachment and rotation forward of the Ciliary Body round an axis represented by the Scleral Ring (×). The Supra-Choroidal Space appears in consequence as an actual, and not as a potential, space; while the Ciliary Muscle bundles and their interstitial tissue present a teased-out appearance, instead of the compact arrangement shown after fixation in Formalin.

The base line connecting the posterior extremity of Schlemm's Canal with the most Anterior and inner Circular bundles (*c*) of the Ciliary Muscle now traverses the Angle instead of falling outside.

The arrow points in the direction of a force of restitution which would convert the conditions existing here to those similar to an eye fixed in Formalin, *vide* Fig. 2.

In effect the detachment is a rotation of the Ciliary

Body round about an axis represented by the Scleral Ring (×), to which the Ciliary Body remains fixed, while its tail, or *pars plana*, is pulled inwards, necessarily causing a rotation *en masse* of the Ciliary Body and Processes. The greater the detachment, the more pronounced is the displacement of the Ciliary Body, and therefore the more distorted becomes the relative position of the structures entering into the formation of the Angle. The detachment and rotation forwards of the Ciliary Body relaxes the Cribriform Ligament (*a*), and likewise causes a rucking of the Iris Root (*d*), whose anterior surface, instead of forming a practically straight line, now forms a knuckle or bend. It is thus that at the circumference of the Anterior Chamber the appearance of a sinus or bay is created. The horns of this bay are composed on the outer side by the slackened ligament (*a*), and on the inner by the similarly rucked anterior surface of the Iris Root (*d*); while the bend of the bay is regarded as being formed by a portion of the Ciliary Body proper. As far as it goes, this description of the Angle as an annular sinus is perfectly correct, but then it only holds good for globes fixed in Müller's Fluid, in which reagent the true shape of the angle is not conserved. The arrow in Fig. 3 points in the direction of a force of restitution which would convert the angle of this Müller-hardened eye to the same form as that presented after fixation in formalin (Fig. 2).

With replacement of the Ciliary Body in position, the base line would be shifted outwards, the slackened Cribriform Ligament pulled upon and straightened, the rucked Iris root smoothed out, and the circumference of the Anterior Chamber restored to its true angular shape.

The topography of the "Filtration Angle" can only be properly studied in eyes in which the Ciliary Body remains *in situ*—*i.e.*, in globes fixed in Formalin (Figs. 2 and 6). The relative position of the structures is then undisturbed, and the appearance presented in sections differs altogether from that sinus formation, on which so much emphasis has been laid as a predisposing cause of Glaucoma, as tending to cut off the circumference from the rest of the Anterior Chamber.

The Sinus shape of the Angle is, in fact, a purely artificial production never found in a Formalin-fixed globe, and therefore it can play no part as a predisposing or accessory factor in Glaucoma.

In this connection it is most instructive to note the difference in the form of the angle in serial section of a Müller's Fluid globe, where the passage outwards of a large anterior ciliary vessel tethers the Ciliary Body to the Sclera and prevents detachment, and to compare the appearance of the angle here with that seen elsewhere in the circumference of the Anterior Chamber where detachment exists.

Where the Ciliary Body is not markedly detached, the shape of the Angle conforms to that in a Formalin-fixed eye; but the farther one gets away from such tethered areas, the more does the appearance become altered, thus showing that the apparent sinus formation of the normal Angle depends directly on the presence of the artificial detachment of the Ciliary Body induced by the fixing reagent.

In Formalin fixed globes detachment is not produced, and the formation of the Angle of the Anterior Chamber is the same all the way round, being constituted on the inner side by the Iris Root, and on the outer by the Cribriform Ligament, the apex being formed by the junction of these two, so that the Ciliary Body proper is altogether excluded (*vide infra*).

Oblique Sectioning.

It will be advisable, before describing the microscopic anatomy of the Cribriform Ligament, to consider some of the technical difficulties in its study which arise from its lattice-work formation and circumferential disposition.

The Cribriform Ligament forms the outer boundary of the Angle of the Anterior Chamber; and being placed circumferentially, its origin, formation, and termination, can only be correctly made out in sections which run truly radially through the globe, a fact accentuated by studying a set of serial sections, in which the farther one gets from a truly meridional or radial section the more obscure become its minute details, owing to the obliquity with which the fibres are sectioned.

That oblique sectioning causes loss of definition of the object under examination has been recognized since the dawn of microscopy, yet this fact has apparently been lost sight of in the case of the eye, although in it obliquity of sectioning is quite unavoidable, owing to the circumferential disposition of its structures.

Heretofore observers have been satisfied in examining only random sections of the eye, oblivious of the fact that, unless the sections run truly radially, fibres are cut obliquely, and therefore their true course cannot be traced, as not only are fibres cut across before they have come to their termination, but also because their exact mode of ending tends to be obscured and blurred by the oblique position in which they lie in the section. The result of not taking these various factors into account is in great part responsible for the description given of the apparent origin of the Pectinate or Cribriform Ligament from a splitting up of Descemet's Membrane. In this, the universally accepted description, no reference is made to the nature of the relationship between the innermost corneal lamellæ and the most anterior and outermost fibres of the Ligament. That the innermost corneal lamellæ are associated with the origin of the Ligament is apparent, for between the point where Descemet's Membrane is described as breaking up into the individual fibres of the Ligament, and the anterior extremity of Schlemm's Canal, there exists an area where the two structures are brought into direct contact. Thus important questions arise which require solution in respect to the nature of this association and the relation of the corneal fibres to the termination of Descemet's Membrane, as well as consideration of possible communication between the corneal interspaces and the trabeculæ of the Ligament. That such questions should not have been formulated is strange, considering that it is well recognized that the deeply-placed infiltration of the cornea in parenchymatous keratitis or in hypopyon ulcer is continuous with a similar infiltration in the network of the Ligament. Such cellular infiltration allows one, even in random sections, to see the connection between the interspaces of the inner corneal lamellæ and the trabeculæ of the Ligament.

In the normal eye such a connection also exists, but it is not so obvious as when each fibre is outlined by infiltration. It requires, in fact, some appreciation of the changes introduced by oblique sectioning to realize why such an association is not always obvious at first sight. When these changes are taken into account, by reconstructing from serial sections the individual course and termination of the innermost lamellæ of the cornea, the very important anatomical relationship is brought out, that the innermost corneal fibres themselves, and not Descemet's Membrane, constitute the origin of the Ligament. This fact I have established, not by random sections, but by overcoming the difficulties of oblique sectioning by serial reconstruction.

Oblique sectioning does not change the actual position of the individual fibres to each other; but it does alter their presentation under the microscope by producing foreshortening or fore-lengthening, besides creating a lack of definition which adds to the difficulty of tracing the fibres to their proper termination.

The effect which obliquity in sectioning causes in the appearance of the retiform structure of the Cribriform Ligament can be better understood if one considers what happens in the case of a hypothetical structure, consisting of parallel rows of fibres of uniform thickness, and having regular interspaces, the fibres and interspaces both measuring, say, 5 micro-millimetres (Fig. 4). If sections are made through this structure of, say, 20 micro-millimetres thickness, which is the average thickness of sections of the eye embedded in celloidin, then the surface or microscopic appear-

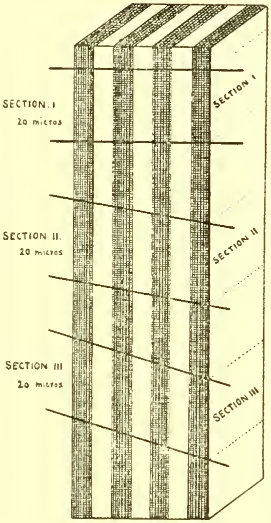

Fig. 4.—Hypothetical Structure sectioned in Different Planes.

The structure is composed of parallel layers of connective tissue, each 5 micro-millimetres thick, separated by interspaces of the same width. The structure is represented as marked out by three sections, each of a thickness of 20 micro-millimetres. The first section runs truly transversely, the second has a slight obliquity, while the third section runs obliquely through the structure. The appearance presented on surface view, as under the microscope, by these various sections varies, depending on the obliquity with which the section goes through the structure. (See Figs. 5, 6, and 7.)

ance of such sections will depend on whether the section runs transversely as Section I., or with slight obliquity as Section II., or with greater obliquity as Section III.

In Fig. 5 the section is one which goes perfectly at right angles through the structure, and therefore it appears microscopically as consisting of sharply-defined and regularly-disposed fibres, separated by equally definite interspaces.

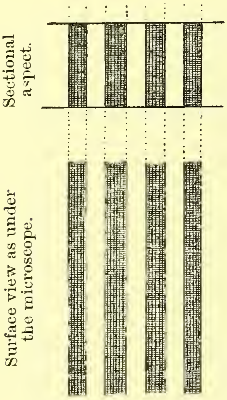

Fig. 5.—Transverse Section.

The upper part of the figure shows, in sectional aspect, Section I. of Fig. 4, represented in the lower part of the diagram in surface or microscopic view, in which the structure appears as composed of sharply-defined bars with clear-cut edges and regular interspaces.

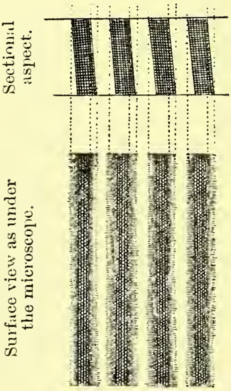

Fig. 6.—Transverse Section with Slight Obliquity.

The section here represents Section II. of Fig. 4, which runs with a slight obliquity, and therefore on sectional aspect the fibres lie with a tilt, which causes on surface view a narrowing of the interspaces; and, as no two adjacent points on the vertical depth of the fibres are properly superimposed, the edges of the fibres appear blurred and indistinct.

Fig. 6 represents a section which runs with a slight obliquity. On sectional aspect the fibres lie somewhat tilted, so that on surface view the individual fibres appear with margins somewhat indistinct and blurred, as no two adjacent spots of the depth of the section lie in the same vertical plane. At the same time the overhanging edges of the obliquely-set fibres make them appear broader by encroaching upon the interspaces, which latter appear proportionately reduced.

Fig. 7 shows an oblique third section in which the fibres are so tilted that on surface view they overlap and altogether obscure the interspaces. The appearance is one of total fogging with loss of all detail, as the slant at which the fibres lie masks the interspaces. Instead of looking vertically through each fibre, one is now viewing their sides; hence the fogged appearance, as identical spots are still farther removed from the vertical plane than in the previous

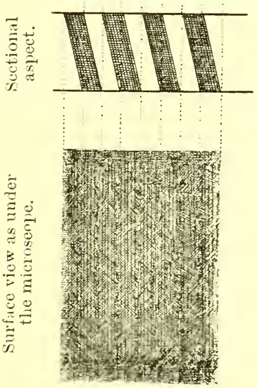

Fig. 7.—Oblique Section.

The oblique Section III. of Fig. 4 lies with its fibres so sloped that, on surface views, the microscopic picture of the structure is altogether obscured. The interspaces are lost to sight, and the whole structure presents a blurred and fogged appearance.

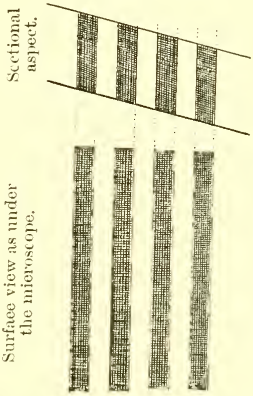

Fig. 8.—Oblique Section placed to correct Obliquity.

The oblique section is here tilted so that the fibres now lie in a vertical plane, and the effect of the obliquity of sectioning is counteracted. The corresponding points in the vertical depth of individual fibres become again superimposed, and the picture produced on surface or microscopic view will be that of sharp, clearly-defined fibres, with regular interspaces, exactly similar to the picture presented by a truly transverse section.

section. If now such a section is tilted (Fig. 8), and examined in the same position as it was made—in other words, if the obliquity of the section is corrected—the fibres will appear in their true and proper perspective.

The interspaces will become manifest, and the margins of the fibres will show up again clearly and sharply defined, just as if the section at the outset had been made truly transversely.

One can very easily demonstrate to oneself these elementary considerations of microscopy by constructing a simple working model on the principles of the hypothetical structure illustrated in Fig. 4, by taking a number of ordinary glass slides and painting on each with Eosin a series of parallel bars to scale. Section I. will be reconstructed by superimposing a sufficient number to build up the thickness of the section. By moving the slides on each other all the various effects of oblique sectioning, already described, can be noted.

Although what we have just been considering respecting the results of oblique sectioning is of a very elementary character, yet unfortunately it has never been sufficiently applied to the histological study of the eye. This lack of recognition of the effect produced by oblique sectioning is responsible for the absence of any precise data respecting the relationship of the inner corneal lamellæ and interspaces to the fibres and trabeculæ of the "Pectinate" Ligament. In the study of this relationship, an appreciation of the above-mentioned principles of microscopy is of essential importance.

There is nothing prettier than viewing under the low power of the microscope a section of the Anterior Segment of the eye, in which, owing to obliquity, the Cribriform or "Pectinate" Ligament appears with the adjacent corneoscleral fibres as diffuse, blurred, and fogged structures, and noting how, as the slide is tilted, so as to correct the obliquity (as in Fig. 8), the parts resolve themselves into their true and proper form. What previously appeared on the flat as a confused mass now shows up as a definite retiform structure composed of sharply-defined fibres, whose trend can be made out with ease. This altered appearance is altogether the result of placing the component elements of the structure in their proper position, which alone allows of clear definition.

In microscopy every advantage lies with definition as against mere magnification; for the higher the power, the greater is the fogging and confusion due to the depth or thickness of the section; and this blurring increases geometrically with magnification.

When any random section of the Cribriform Ligament is so tilted on a levelling stage as to counteract oblique sectioning, then the origin, course, and termination of its individual fibres can be clearly defined just as if one were looking at a truly radial section.

This is a point to be emphasized, because if due consideration is given to the results produced by oblique sectioning, and the means by which such results can be counteracted, it will be found, as will be presently demonstrated, that the origin and termination of the fibres composing the network on the inner side of Schlemm's Canal differ materially from their ordinary textbook description.

The Cribriform Ligament.

Having discussed the effect of artificial detachment of the Ciliary Body in altering the configuration of the Angle of the Anterior Chamber, and noted the difficulties introduced by its circumferential disposition, we are in a better position to approach the examination of the formation and interrelationship of its constituent elements.

The description of these structures which follows is based on a study of serial sections of globes fixed in Formalin, the results of which have been controlled at every point by a similar examination and reconstruction of serial sections of eyes fixed in Müller's Fluid.

I have in all sectioned and mounted in series over five thousand consecutive sections of both normal and pathological globes, including one series, consisting of over six hundred sections, which comprised more than half the circumference of a normal eye.

By following out this method of research, errors arising from hasty conclusions, the result of random and therefore imperfect sections, are eliminated, as it is only by piecing together all the various consecutive sections that the final and completed picture is produced. At the same time the difficulties introduced by unavoidable obliquity of sectioning are much more easily noted, and counteracted. The data obtained by reconstructing serial sections are so opposed to current teaching that it is imperative at the outset to clearly realize the basis on which is founded the description which follows of the structures composing the Angle.

The first point brought out by serial reconstruction is the inappropriateness of the term *Ligamentum Pectinatum Iridis.* This process demonstrates that neither is this netlike structure the comblike formation that its name implies, nor do its fibres terminate in or act as a Ligament to the Iris, as described in textbooks. To dispel, therefore, all the wrong and misleading associations of the old terminology, I have suggested in its place the term Cribriform Ligament.

The Cribriform Ligament is a structure which in its origin, formation, and termination, is altogether different from what is implied by the term "Pectinate" Ligament, and hence comes the necessity of a new designation. The Cribriform Ligament arises, not by a splitting up of Descemet's Membrane, but as a continuation of the inner Corneal Lamellæ; while its structure is formed of regular and evenly disposed interlacing fibres, and not composed as the Pectinate

Ligament is described, of longitudinal fibres on the inner side, and circular fibres on the outer. The final point of distinction which requires emphasis is that the Cribriform Ligament ends not in the Iris Root, but as the Ligament of origin for the Ciliary Muscle (Fig. 9).

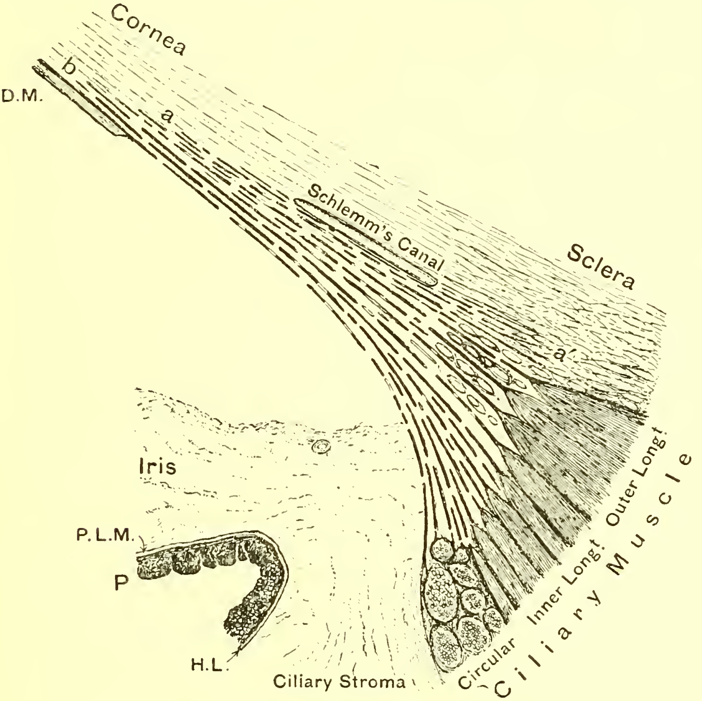

Fig. 9.—The Cribriform Ligament.

The inner lamella of the Cornea (*a* and *b*) are continuous with the fibres of the Cribriform Ligament. The outer fibres, which start from *a*, lie next to Schlemm's Canal, and terminate at *a'* in the scleral fibres. The inner fibres, which start from *b*, spread out in a fan-shaped manner to act as fibres of origin to the longitudinal muscle bundles, and as check fibres to the circular bundles of the Ciliary Muscle. The pigment epithelium (P.) and posterior limiting membrane (P.L.M.) of Iris are continuous with the respective pigment and hyaline layers (H.L.) of the Ciliary Body.

Descemet's Membrane (D.M.).

The structure forming the outer boundary of the Angle of the Anterior Chamber is, in short, not a comblike or

Pectinate Ligament for the Iris Root, but the perforated or Cribriform Ligament of the Ciliary Muscle. A change in nomenclature is therefore essential. The term Cribriform Ligament is in every way a suitable one, being in absolute conformity with both the anatomical and physiological characters of the structure, while at the same time it is one which facilitates the description and simplifies the conception of the " Filtration " Angle.

A factor of great value and assistance in making out the disposition of the Cribriform Ligament is the progressive histological alteration which this structure undergoes with increasing years.

At birth the Cribriform Ligament is a purely cellular structure indistinguishable posteriorly from the cellular formation of the Iris and Ciliary Body, but which elsewhere stands out in marked contrast to the surrounding fibrous structure of the corneo-sclera. With each advancing decade the trabeculæ of the open network of the Cribriform Ligament manifest more and more fibrous changes, so that finally, in a senile eye, the histological contrast between the structure of the Filtration network and the surrounding sclera is reduced simply to a difference in the size of the connective-tissue fibres; while a correspondingly sharp distinction is created between the connective-tissue fibres of the Ligament and the cellular stroma of the Iris and Ciliary Body. This physiological sclerosis of the Cribriform Ligament simplifies very much the tracing of its origin, formation, and termination ; for by employing such selective methods as Van Gieson's connective-tissue stain, a sharp contrast is obtained of the utmost value in defining the course of the individual and now sclerosed fibres, and in tracing their origin from the inner corneal lamellæ, and their termination into the Ciliary Muscle.

The comparison of meridional sections through the angle of the Anterior Chamber, with transverse and tangential series, confirms in every particular the appearance presented by each one of the series. The principle of the arrangement of the fibres composing the Cribriform Ligament is fundamentally very simple, as it corresponds exactly to that which obtains in the fibres of the immediately surrounding tissue. The Cribriform Ligament is thus nothing more than a regular and open network, formed of interlacing fibres, which are a direct continuation of the longitudinal and circular bundles of the sclera about the venous sinus of Schlemm's Canal.

The description given of the apparent origin of the Cribriform Ligament, as derived from a splitting up of Descemet's Membrane, is not borne out by serial sections. The apparent origin is an artificial one depending on oblique sectioning, and is proved to be so by examining any random section on a levelling stage, and correcting for obliquity as already described (see Fig. 8).

On an ordinary microscope stage one will get approximately the same results by simply tilting the slide by hand either on its long or short axis. Even in this rough-and-ready way one will be able to note a most marked difference in definition of the individual fibres of the Ligament.

In any meridional section through the Angle all the structures which go to form the Cribriform Ligament are bound to be present; but unless a true radial section has been cut, any obliquity will produce not only a lack of definition of the individual fibres, but also, as some fibres will be cut short before they reach their termination, a false perspective owing to fore-shortening or fore-lengthening of fibres. In this way an imperfect picture of the true state of affairs is produced.

The apparent splitting up of Descemet's Membrane into the Cribriform Ligament arises from a false perspective produced by unavoidable obliquity in consequence of the circumferential disposition of the Angle. On this account, even if one should happen to cut an ideal and perfectly radial section, the next section made would theoretically not be truly radial, and the third still less so, and so on, each section manifesting more and more obliquity. The difficulty of obtaining radial sections emphasizes the importance of serial reconstruction, as well as the value of studying individual sections by examining them on a levelling stage, so that the faulty appearances and lack of definition induced by oblique sectioning may be overcome.

The Origin of the Cribriform Ligament.

The origin of the Cribriform Ligament can be made out with certainty and ease in sections of a senile eye stained after Van Gieson's method. In such eyes the fibres composing the network are sclerosed, and therefore the only histological difference between them and the corneal fibres is one of size. As Descemet's Membrane does not stain the same rich red colour as connective tissue, it stands out under such circumstances quite apart and distinct from the Ligament, whose sclerosed fibres are thus easily traced as a direct continuation of the inner corneal lamellæ (Fig. 9).

The corneal lamellæ, next to Descemet's Membrane, break up into the innermost fibres of the Cribriform Ligament at a point (*b*) just anterior to the termination of the Membrane. The superimposed lamellæ project farther back before splitting up (*a*), so that in a meridional section the Anterior Extremity of the Cribriform Ligament produces a rough and general impression of an acute-angled triangle with apex forward (see Fig. 2). The inner limb is sharply defined, and constitutes the outer boundary of the Angle of the Anterior Chamber. The outer limb of the triangle is formed by the line along which the inner corneal fibres break up into those of the Ligament, and therefore is not sharply defined; for it is important to note that it is in no sense an anatomical line, but purely a physiological one, which marks the cleavage of the corneal fibres into those of the Cribriform Ligament. Anatomically there is complete continuity between the one set of fibres and the other, a fact which can only be made out with comparative ease in senile eyes in which the Cribriform fibres are sclerosed. In animals' eyes, as in the pig, dog, and cat, the continuation of the one tissue into the other can be defined with much greater ease, as in them there exists a considerable amount of pigmentation normally present about the region of Schlemm's Canal; so that pigment masses in the trabeculæ of the Ligament can be seen to be continuous with similar pigment masses lying in the interstitial spaces of the inner corneal lamellæ, a clear proof not merely of the corneal origin of the Ligament, but also that the alveoli of the Ligament must be continuous with the lymph spaces of the cornea and their contained fixed corpuscles.

The anatomical continuity of the inner corneal lamellæ and interspaces with the fibres and spaces of the Cribriform Ligament brings about a significant association between the Aqueous in the Anterior Chamber on the one hand, and the Corneal tissue on the other, an association the physiological importance of which must be deferred till later.

Formation and Termination of the Cribriform Ligament.

The Cribriform Ligament is composed of circular and longitudinal fibres, which enter in equal and similar proportions into the composition of its whole structure; in other words, the outer portion is not exclusively composed of circular fibres, or the inner of meridional lamellæ. The apparent disproportion seen in meridional sections between the two sets of fibres is purely artificial, as is shown by a comparison of serial sections made in different planes through the region of Schlemm's Canal in the same specimen. Such a comparison shows that the circular fibres entering into the construction of the Cribriform Ligament are best made out in tangential or transverse sections, while the longitudinal fibres are best seen in meridional ones.

The Circular Fibres entering into the formation of the Cribriform Ligament are associated and continuous with the circularly-disposed connective-tissue bundles surrounding Schlemm's Canal. These circular fibres assist in binding the whole structure of the Ligament together, as well as helping to fix it down in its position. The region of the corneo-sclera contains, as is well known, a large number of fibres running in a circular direction, of which the most prominent collection lies just posterior to Schlemm's Canal, where it has received the name of the "Scleral Ring" (Fig. 10, S.R.).

This particular aggregation of circular fibres is of great importance, as will be noted later, in keeping the Ciliary Muscle in position.

The Longitudinal Fibres of the Cribriform Ligament are not derived from a splitting up of Descemet's Membrane, the cause for the appearance of which I have already discussed, but they exist as a direct continuation backwards of the innermost lamellæ of the cornea (Fig. 9).

Traced backwards, these longitudinal fibres can be divided according to their course and termination into—

1. A small outer or scleral division (*a′*).
2. A large muscular or ciliary division (*b′*).

1. *The Outer or Scleral Division* comprises only a small part of the Ligament, which runs backwards and outwards next to Schlemm's Canal, and at the posterior extremity of that venous sinus its fibres become incorporated with and lost in those of the sclera (*a′*).

2. *The Inner or Ciliary Muscle Fibres* spread out in a

fan-shaped manner to act as the Ligament of attachment for the Ciliary Muscle.

In structure the Cribriform Ligament is thus nothing more than an open part of the Corneo-scleral envelope, and as such it consists of longitudinal and circular fibres arranged crossways as a warp and a woof, extending as

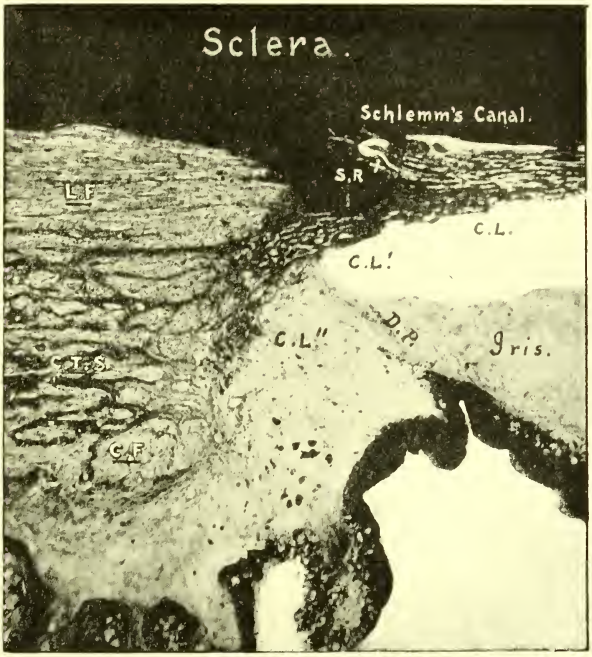

Fig. 10.—Micro-Photograph of the Cribriform Ligament.

A man, aged sixty years. Eye fixed in formalin. Van Gieson's connective-tissue stain. There is marked and complete sclerosis of the fibres composing the cribriform ligament (C.L.). At C.L.' the fibres lie on the inner side of the scleral ring (S.R.), and terminate (C.L.") by being incorporated into the connective-tissue stroma (C.T.S.), here markedly sclerosed, of the circular fibres (C.F.) of the ciliary muscle.

L.F., Longitudinal fibres of the ciliary muscle. D.P., Fibres of origin of a fasciculus of the musculus dilator pupillæ. ×, Posterior extremity of Schlemm's canal, into which opens a direct tributary from the ciliary venous plexus which has made its way along the fibres of the cribriform ligament internal to the scleral ring. *Vide* Fig. 34 (2).

an open network across the inner side of Schlemm's Canal from the termination of the inner lamellæ of the Cornea to the Ciliary Muscle bundles.

The Cribriform Ligament, as the true ligament of origin of the Ciliary Muscle, is pulled taut, and its alveoli opened to their fullest extent, when the muscle is thrown into action.

The loose connective-tissue stroma of the Iris Root is attached to the fibres of the ligament at a point just posterior to the Scleral Ring. The Ciliary Body proper is thus excluded from taking any part whatever in the formation of the Angle of the Anterior Chamber, which is a true angle, the outer boundary being formed by the Cribriform Ligament, and the inner by the anterior surface of the Iris Root, while its apex is formed by the junction of these two.

This attachment of the Iris stroma to the Cribriform Ligament is quite a different arrangement from that described in all text-books where the fibres of the ligament are represented as bending into the Iris Root, which they certainly do not.

This apparent termination is the result of several factors, of which the most important are oblique sectioning and displacement forward of the circular fibres of the Ciliary Muscle consequent to fixation in Müller's fluid (see Fig. 2).

A combination of these two factors gives a false perspective to the course of the cribriform fibres, thus emphasizing the necessity of having the Ciliary Body in position, as well as the importance of studying serial sections, especially in senile eyes, in which sclerosis of the ligament causes the fibres to stand out in marked contrast to the loose Iris Stroma (Fig. 10).

The Ciliary Muscle.

The general appearance and position of the Ciliary Muscle depends very much on the fixing agent employed. After fixation in Müller's fluid the circular fibres are displaced forwards (Fig. 2), but besides this general displacement the fibres of the longitudinal muscle bundles appear as if they had been teased out, in comparison with the compact and closely-arranged fasciculi existing after the use of formalin (Fig. 10).

Owing to the progressive sclerosis of the Cribriform Ligament, as well as of the interstitial tissue of the Ciliary Muscle, the exact mode of origin of the muscle fibres is best studied in senile eyes in which serial reconstruction shows the perfect regularity and orderliness of the attachment of the muscular fasciculi to their ligament of origin, such as is impossible to obtain by random sections.

The Ciliary Muscle is composed of two well-recognized divisions of Longitudinal and Circular fibres. The fasciculi of the longitudinal fibres are arranged in superimposed layers, while the circular bundles form a true Ciliary Sphincter, which caps the most prominent portion of the muscular division of the Ciliary Body.

The appearance shown in Fig. 9 has been obtained by reconstructing, section by section, the individual course of the muscle bundles in a formalin-fixed globe. The longitudinal fasciculi of the Ciliary Muscle are then seen to be disposed in regular tiers, about ten in number, so arranged that the largest and longest muscle bundles lie to the outer side, while the succeeding internal fasciculi become progressively smaller and subdivide earlier.

The tapering ends of the outer and larger three or four longitudinal muscle bundles are attached to those fibres of the Cribriform Ligament which pierce the Scleral Ring (*b′*), while the smaller and more internally placed fasciculi are attached to those fibres of the ligament which pass internal to the scleral ring (*b″*). The scleral ring itself is not, as has been described, the point of attachment of the Ciliary Muscle, but it merely acts as a fixed point which steadies the Cribriform Ligament during action of the muscle.

Besides a disparity in size between the outer and inner longitudinal muscle bundles, there is another well-marked difference between them in the amount of their interstitial connective tissue.

The outer three or four longitudinal fasciculi possess very little—in fact, hardly any—connective-tissue stroma, while the internal subjacent tiers of muscle bundles possess a very marked interstitial stroma, which becomes more conspicuous the older the eye (Fig. 10, C.T.S.).

The circular fibres of the Ciliary Muscle constitute a true Ciliary Sphincter intimately connected by their interstitial tissue with the most internal fibres of the Cribriform Ligament, which fibres play the part of a check ligament to the circularly-disposed muscle bundles.

Anatomically the Cribriform Ligament, with the exception of the outer portion incorporated in the Sclera, is exclusively associated with the Ciliary Muscle by acting as a ligament of origin to its longitudinal fibres, and as a check ligament to its circular bundles.

When the Ciliary Muscle is thrown into action, the network of the Cribriform Ligament is pulled upon and opened to its fullest extent, excepting of course the outer or Scleral part (*a*, *a′*), which by remaining uninfluenced obviates any pressure being exerted on the walls of Schlemm's Canal during action of the muscle.

The Supra-Choroidal Space.

The Supra-Choroidal Space lymph space is regarded as being closed anteriorly by the attachment of the Ciliary

Body to the Sclera, while such fluid as it contains, as first demonstrated by Schwalbe,* passes by way of the perivascular sheaths of the vortex veins into Tenon's Capsule. Parsons† states that "the lymph which passes by this route is small in quantity, and is derived solely from the Choroid and Ciliary Body, involving only the internal economy of these structures, and having nothing to do with the maintenance of the intra-ocular pressures." This sentence sums up the current physiological views respecting the origin and purpose of the fluid in the Supra-Choroidal Space and the relatively unimportant function of that lymph space, though, in the light of what has been stated regarding the termination of the Cribriform Ligament, these views require considerable modifications.

The open web of the Cribriform Ligament, while allowing the Aqueous free access to the venous sinus of Schlemm's Canal, creates also a route into the Supra-Choroidal Space, through that portion of its network which proceeds backwards and inwards to the circular fibres of the Ciliary Muscle (Figs. 9 and 10).

The only barrier interposed between the Angle of the Aqueous Chamber on the one hand, and the Supra-Choroidal Space on the other, is the insertion of the longitudinal fibres of the Ciliary Muscle, along and between which the Aqueous readily finds its way.

The patency of this communication can be demonstrated experimentally as well as by examination of pathological specimens. After the injection of Berlin Blue or Indian Ink into the Anterior Chamber of freshly excised eyes, I have been able to trace in serial sections the particles of injected material making their way backwards into the Supra-Choroidal Space between the longitudinal fasciculi of the Ciliary Muscle, and that not only in animals' eyes, but also in a freshly excised normal human eye (Fig. 22, P). An equally striking proof of the free communication between these two lymph spaces is to be obtained, however, in cases of chronic irido-cyclitis, in which the uveal pigment has made its way into the Anterior Chamber, and can be traced passing out by this route. Nature's injection experiments are very much finer and more accurate ones than our own crude imitations, which are apt to defeat their own ends by causing clogging of the excretory apparatus.

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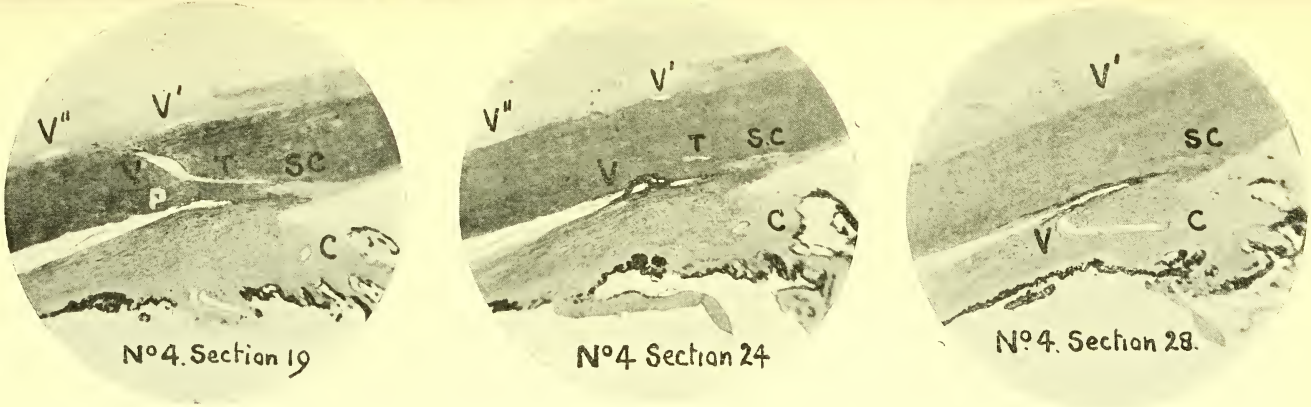
* Schwalbe, "Anatomie des Sinnesorgane."

† Parsons, *loc. cit.*, p. 995.

In tracing out this connection, it is obvious that individual sections cannot in themselves carry any weight, but it is quite otherwise when sections are examined in series (Figs. 11, 12, and 13). Studied in this way, a most striking picture is produced, as then each pigment mass is seen to be, not an isolated unit, but a link in the chain which connects these two lymph spaces together. That the uveal pigment in such pathological cases has made its way into the Supra-Choroidal Space through the open network of the Cribriform Ligament is not only thus proved, but any idea of its having made its way direct from the Pigment Epithelium across the Ciliary Body is at the same time discountenanced.

Figs. 11, 12, and 13, are micro-photographs of sections numbering respectively 19, 24, and 28, of a series from a case of Irido-Cyclitis with marked Keratitis Punctata following extraction. In the various consecutive sections uveal pigment masses can be traced directly connecting the Angle of the Aqueous Chamber with the deeply pigmented anterior extremity of the Supra-Choroidal Space. With the exception of this region, the Ciliary Body is otherwise free of pigment, which would not be the case if the uveal pigment had made its way direct from the Ciliary Epithelium. The only route by which the pigment could have found its way into the Supra-Choroidal Space in this case, as well as in all similar ones which I have examined,



Figs. 11, 12, 13.—Micro-Photographs of Adjacent Serial Sections from a Case of Irido-Cyclitis following Extraction, illustrating the Route by which the Uveal Pigment has obtained Access to the Supra-Choroidal Space.

Fig. 11 (Section 19) shows Schlemm's Canal receiving a tributary from the Vein (V) cut in longitudinal section, the perivascular sheath of which contains pigment granules. The Circulus Iridis Major (C) is shown in cross-section. The anterior extremity of the Supra Choroidal Space contains masses of pigment.

Fig. 12 (Section 24) shows the large vein of the previous section crossing the Supra-Choroidal Space, and surrounded by a thick mantle of pigment. The Circulus Iridis Major (C) has moved slightly outwards.

Fig. 13 (Section 28) shows that the Vein (V) and the Circulus Iridis Major (C), seen in the two previous sections, are one and the same vessel. The Supra-Choroidal Space contains much uveal pigment, but the perivascular sheath of the longitudinally sectioned vessel is devoid of all pigmentation. If the pigment present had made its way directly across the Ciliary Body, then this part of the vessel would have been surrounded by a similar pigment mantle to that which envelops it when crossing the Supra-Choroidal Space.

Individually these sections prove nothing, but collectively they demonstrate that, as the Ciliary Body is quite free from uveal pigmentation, excepting between the Anterior extremity of the Supra-Choroidal Space and the Angle of the Anterior Chamber, the pigment could only have passed in by this route.

is through the posterior portion of the Cribriform Ligament, and then between the fasciculi of the longitudinal fibres of the Ciliary Muscle. This pigmentation indicates that a current of Aqueous flows from the Angle into the Supra-Choroidal Space, and therefore that the fluid in this Space is not a special creation, but is derived simply from the Aqueous. This association between the two lymph spaces is one of great physiological significance, as owing to it the hydrostatic pressure in the Supra-Choroidal Space is always kept the same as that in the Aqueous Chamber. The pressures of Aqueous, Vitreous, and such fluid as is present in the Supra-Choroidal Space, all stand at the same level, and therefore the intra-ocular pressure is transmitted equally on both sides of the Retina; in other words, the hydrostatic pressure on the outer aspect of the Retina balances that on its inner surface, and likewise permeates its whole structure, thereby obviating the serious consequences which would naturally follow if the latter structure were called upon to support any difference of pressure on its two sides.

The Supra-Choroidal Space is thus, from its position and connection with the Anterior Chamber, analogous to the Sub-arachnoid Space of the brain, which by its connections with the lateral ventricles on one hand, and the Cisternæ Sub-arachnoidales on the other, likewise ensures the even distribution of the intra-cranial pressure throughout the cranial contents (Fig. 1).

The Cribriform Ligament in Accommodation.

The physiological importance of the communication between the Aqueous Chamber and the Supra-Choroidal Space is still further emphasized when we consider the relation of the Cribriform Ligament to the Ciliary Muscle, and the effect that action of the Muscle must have in opening its meshes and facilitating the passage of aqueous to both Schlemm's Canal and the Supra-Choroidal Space.

Helmholtz held that as the Aqueous, like all other fluids, is incompressible, therefore the advancement of the lens in accommodation must be compensated for by an anatomical deepening of the Angle, a deepening which has always been assumed and illustrated in textbooks as taking place.

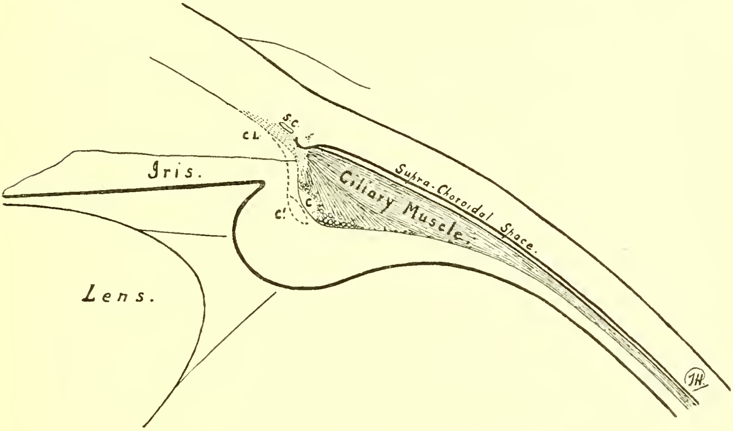


Fig. 14.—The Angle in Accommodation.

The innermost fibres of the Cribriform Ligament (C.L.) terminate in the connective-tissue stroma of the Circular Muscle bundles of the Ciliary Muscle (C), and so play the part of a check ligament.

In accommodation the "Ciliary Sphincter" moves inwards from C to C′, and, the Cribriform Ligament being pulled taut, its meshes will be opened. The Angle of the Anterior Chamber will therefore not be deepened in an anatomical sense, but only in a physiological one by the ready passage of Aqueous into the Supra-Choroidal Space, whereby the volume displaced by changes in position of the Lens and Ciliary Processes automatically finds room for itself.

The mechanism conceived in support of this assumption is based on the idea of a sinus formation of the Angle, and of the existence of such a mode of attachment of the Ciliary Muscle that when thrown into action the Angle would be pulled back and the sinus deepened.

Now, as has already been discussed, the Angle of the

Anterior Chamber is a true angle, and not a sinus, and its outer boundary is formed by the Cribriform Ligament, which acts as the ligament of origin for the longitudinal muscle bundles, while its most internal fibres act as a check ligament to the circular bundles of the Ciliary Muscle, so that it is impossible to imagine how deepening can take place (Fig. 14).

The "Ciliary Sphincter," as the circular fibres of the Ciliary Muscle may well be called, will in accommodation move inwards in a sphincter-like manner from C to C′, and consequently the innermost fibres of the Cribriform Ligament will be pulled upon and straightened out.

Under the anatomical conditions which I have described, the Cribriform Ligament cannot be pulled backwards to cause an anatomical deepening of the Angle, but it is pulled inwards and so becomes taut, causing thereby a physiological deepening by facilitating the passage of aqueous into the Supra-Choroidal Space.

As in accommodation the Ciliary Processes move inwards, they displace a volume of fluid in the Posterior Chamber, which volume, as well as the Aqueous volume displaced by the advancing lens, finds room by a facilitated access into the Supra-Choroidal Space.

It is by means of such a self-regulating mechanism that the act of accommodation is prevented from influencing the intra-ocular pressure or disturbing the balance of pressure between the outer and inner aspects of the Retina (*vide infra*).

Sclerosis of the Cribriform Ligament.

The Cribriform Ligament undergoes throughout life a progressive and physiological sclerosis, so that from a cellular structure at birth it becomes a purely fibrous formation in old age.

The sclerosis first starts in the inner or Ciliary division of the Ligament, being induced, no doubt, by the constant traction of the Ciliary Muscle on its ligament of attachment.

The outer or scleral portion, not being influenced by muscular action, is not subject to fibrotic changes till very much later in life.

In consequence of the simple and regular lattice-work arrangement of the Cribriform Ligament, and of the slowness of the process of fibrosis which it undergoes, we possess in it an ideal structure for the investigation of the formation and development of connective and elastic tissue.

This physiological process of fibrosis is steadily progressive, and therefore more easy to study; but much more valuable and important than this is the fact that each individual fibre of the Ligament is isolated and distinct from its neighbour, so that the development of fibrous tissue can be studied in discrete fibres, and not merely in a compact tissue. The Cribriform Ligament is formed by columns of cells arranged in regular rows, with a fixed interval between each column, the whole undergoing a slow fibrosis from early childhood onwards; so that in a series of eyes, by taking a particular strand, say the innermost bounding the angle of the Anterior Chamber, one can trace step by step the various changes that precede and culminate in connective-tissue formation.

At birth the Cribriform Ligament is a purely cellular structure composed of regularly-arranged strands of spindle-shaped cells (Fig 15, A).

Before long, however, the individual columns of cells develop round their respective centres, by a process of secretion or excretion, a homogeneous substance similar to that composing Descemet's Membrane (Fig. 15, B). This in a measure accounts for the accepted description that Descemet's Membrane splits up at its periphery to form the Cribriform Ligament.

About the fifth year—that is, about the time when the Cornea reaches its full dimensions—the strands composing the Ciliary division of the Ligament begin to manifest fibrous changes. This fibrosis, seen best in sections stained by Van Gieson's stain, begins *in the centre* of the homogeneous strands. The appearance now is that of a central core of

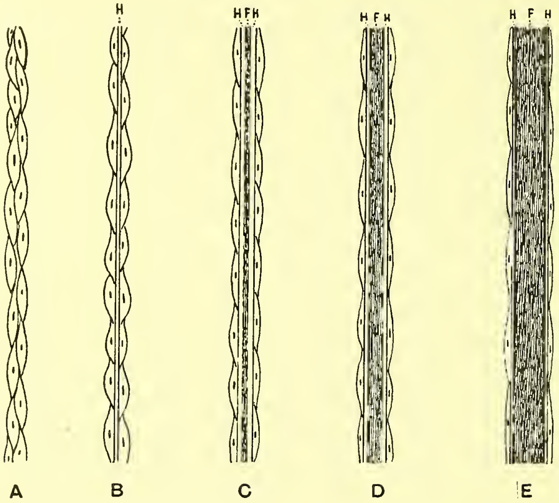


Fig. 15. — Diagrammatic Representation of the Process of Connective-Tissue Formation in the Strands of the Cribriform Ligament.

A. At birth the ligament is composed of regularly-arranged strands of spindle-shaped cells.

B. Soon after birth these cellular columns proceed to form round their respective centres a homogeneous substance (H), similar in nature to that of Descemet's Membrane.

C. By the continued activity of the cells this central formation increases in thickness, and about the fifth year fibrous changes become manifest in the core of the homogeneous matrix. The appearance is that of a filament of fibrous tissue (F) embedded in a thick envelope of homogeneous substance (H).

D. As the strands grow in thickness, they become more and more fibrous, the homogeneous envelope (H) less marked, and the cellular less numerous.

E. Finally the strands are totally converted into thick connective-tissue fibres (F), enclosed in merely a shell of a homogeneous matrix (H) ; the covering cells become still more reduced in number.

connective tissue, enclosed in a thick envelope of homogeneous matrix, similar to that composing Descemet's Membrane, the whole column being surrounded by endothelial cells (Fig. 15, C).

With each advancing decade more and more homogeneous material is laid down by the covering cells, and is with equal rapidity converted from the centre into fibrous tissue, so that the thickness of the strand becomes increased (Fig. 15, D), till finally the whole column is converted into a solid connective-tissue fibre with only the thinnest envelope of a homogeneous matrix (Fig. 15, E). On the outside, and lining the alveoli, lie the cellular elements from which these formations were derived, which elements, as age advances and sclerosis increases, become much reduced in number.

The early fibrosis in the inner or Ciliary division of the Cribriform Ligament is undoubtedly determined by the action of the Ciliary Muscle, to which this portion of the Filtration Network acts as a ligament of origin.

The outer portion of the Cribriform Ligament, being lost posteriorly in the Sclera, remains unaffected by the action of the Ciliary Muscle, and therefore fibrosis here is of very much later development, and is determined by the same agencies which, associated with advancing years, induce sclerosis elsewhere in the body. Although of much later origin, occurring about and after middle life, the process of fibrosis follows exactly the same line of development as that met with in the inner division of the Ligament.

It would subserve no practical purpose to discuss here the question of the distribution of the elastic tissue entering into the composition of the Cribriform Ligament—its development in youth, distribution in adult life, and disappearance in old age. Suffice it to say that a study of the progressive alterations that take place throughout life in the structure of the Ligament demonstrates the similarity of the extra-cellular development from the same elementary cells in the case of both elastic and connective tissue.

The development of these tissues does not proceed from a direct conversion of the cell protoplasm, but both tissues are formed indirectly by the alteration and transformation of a substance which is itself, however, the product of cellular activity.

The histological result of the total sclerosis of the Cribriform Ligament is to reduce the contrast between it and the surrounding corneo-sclera solely to one of the magnitude of the fibres.

The anatomical result of the sclerosis of the Ligament is greatly to reduce the interspaces and alveoli of the retiform structure, and thus seriously to impede the ready access of the Aqueous to Schlemm's Canal.

The depth or thickness of the Cribriform Ligament at a point corresponding to the middle of Schlemm's Canal is throughout life about 0·1 millimetre. In a child of five years of age, when sclerosis is just commencing, the diameter of the individual strands of the Ciliary portion of the ligament is about 3 micros. (Fig. 15, C).

The central core of fibrous tissue measures only 1 micro. in thickness, while its enclosing envelope, structurally similar to Descemet's Membrane, surrounds the connective-tissue strand with a sheath also 1 micro. in thickness, thus making the total diameter of the strand 3 micros. exclusive of cellular elements. In a man of fifty, the total diameter of each ciliary strand of the ligament is generally about double that in a child of five, being from 5 to 7 micros. (Fig. 15, D and E).

The diameter of the central core of connective tissue increases from 1 micro. to 4 or 5 micros., while the thickness of the non-fibrous envelope is reduced to a negligible quantity.

As the space between Schlemm's Canal and the Anterior

Chamber is the same in a man of fifty as in a child of five, it is obvious that the increased size of the individual strands, amounting to twice their former thickness, will cause in the older eye a marked diminution in the lumen of the alveoli of the perforated ligament.

This occlusion of the meshes of the Cribriform Ligament, by obstructing the passage of the Aqueous to Schlemm's Canal, predisposes the presbyopic eye to Glaucoma.

When the whole structure of the Cribriform Ligament is reduced to a fibrous reticulum, it is easy to make out in serial sections that this Network is not a derivative of Descemet's Membrane, but a direct continuation of the inner Corneal lamellæ, and, further, that the alveoli formed by the sclerosed strands are continuous with the lymph spaces of the cornea and their contained fixed corpuscles, thus bringing the latter into direct association with the endothelial cells of the Anterior Chamber.

This anatomical continuity has a considerable bearing, not only on the question of the nutrition of the cornea, but also in accounting for the clinical course and pathological findings of deep-seated corneal affections.

The Corneal Periphery.

The cornea appears to the naked eye as affixed to the sclera like a watch-glass, to use an oft-quoted simile, but histologically there is no such clearly defined distinction, as both tissues react similarly to staining reagents, and the one set of fibres passes imperceptibly into the other.

The cornea is associated not merely embryologically, but also anatomically, with the adjacent tissues of the conjunctiva and uvea. The innermost corneal fibres break up at the circumference of the Anterior Chamber into the retiform structure of the Cribriform Ligament, and therefore these lamellæ, along with Descemet's Membrane and its endothelium, must be classed together as constituting the Uveal portion of the Cornea—an anatomical relationship which is in complete accord with Waldeyer's embryological classification of the Corneal layers.

While the middle lamellæ of the Cornea are interwoven with the Scleral fibres, the Anterior or outermost fibres next to Bowman's Membrane break up and terminate in a feathery manner in the tissue of the Limbus Conjunctivæ (Fig. 16). To make out this mode of termination of the superficial lamellæ of the Cornea, it is necessary that the same means be taken to overcome and correct the appearance produced by oblique sectioning, as in the case of the inner lamellæ and the Cribriform Ligament, for the two conditions are exactly analogous.

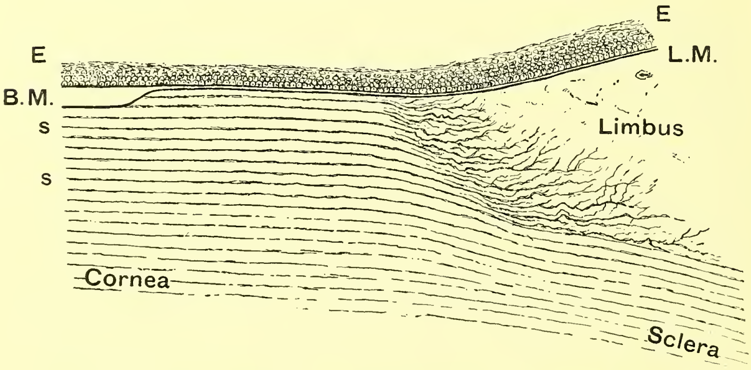


FIG. 16.—TERMINATION OF THE SUPERFICIAL CORNEAL FIBRES.

The superficial corneal fibres (S, S) break up into a number of fine fibres, which are lost in the loose stroma of the *Limbus conjunctivæ.* Bowman's Membrane (B.M.) is continuous with Limiting Membrane (L.M.) of the conjunctival epithelium.

The Superficial Corneal Lamellæ split up as they reach the Limbus into a number of fine fibrillæ, which become lost in the loose stroma of the Sub-Conjunctiva. A close connection is thus created between the Limbus Conjunctivæ and the Superficial layers of the cornea, the physiological importance of which will be discussed later.

Bowman's Membrane terminates by becoming continuous with the Limiting Membrane of the Conjunctival epithelium, but both in aspect and position its ending is variable; for while as a rule it ends abruptly at or within 0·3 millimetre of the Limbus, at other times it gradually fades into the Limiting Membrane.

The peripheral termination of the outer Corneal fibres in the Limbus, and of the deeper ones in the Cribriform Ligament, associates the external Lamellæ with the Conjunctiva, and the internal with the Ciliary Muscle and Uvea. These anatomical associations support Waldeyer's views* as to the development of the Cornea (Fig. 17).

According to Schwalbe's classification,† which is the one most generally accepted, the Cornea is constituted of the three following divisions:

1. A Conjunctival Cornea formed by the Epithelium alone;
2. A Scleral Cornea, which includes Bowman's Membrane and the whole of the Corneal tissue; and
3. An Uveal Cornea, consisting of Descemet's Membrane and the Internal Endothelium.

My own anatomical observations, which have not been confined merely to the human eye, agree with and strongly support Waldeyer's embryological classification of the layers of the Cornea as consisting of—

1. A conjunctival or outer stratum, comprising the surface epithelium, Bowman's Membrane, and those outermost lamellæ of the Cornea which terminate in the Limbus.
2. A Scleral or Middle Layer.
3. An Uveal or Inner Portion, consisting of the posterior

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* Waldeyer in "Graefe-Saemish Handbuch," 1st edit., vol. i.

† Schwalbe, "Sinnesorgane," 1887.

corneal endothelium and its derivative Descemet's Membrane, and those innermost lamellæ of the Cornea which peripherally split up to form the Cribriform Ligament.

In connection with this classification it is interesting to note that the increased thickness of the Cornea, as compared with the Sclera, is due in great part to the addition to its thickness of the Conjunctival and Uveal Strata. The Sclera just posterior to the Corneo-Scleral junction has a thickness of 0·6 millimetre, while the Cornea at its periphery measures about 1 millimetre. This extra thickness is due to the Outer Stratum (Epithelium and outermost Corneal Lamellæ) adding about 0·2 millimetre, and the Innermost Stratum about 0·1 millimetre, to the original thickness of the Middle Scleral layer of the Cornea.

Comparative Anatomy of the Angle of the Anterior Chamber.

The comparative anatomy of the Angle of the Anterior Chamber is in perfect conformity with the description already given of its structure and formation in the human eye—at any rate, in the case of all those animals which I have personally examined, and they include such a representative class of the mammalia as the monkey, sheep, pig, dog, cat, and rabbit.

In man I have described the Angle of the Anterior Chamber as a true Angle formed by the attachment of the Iris to the Cribriform Ligament, and the same arrangement is met with in the lower animals, with this difference, that in them the Iris Root is broken up into a series of pillars which are attached to the Cribriform Ligament, in some cases as far forward as Schlemm's Canal (Fig. 17). Serial sections show that the Cribriform Ligament serves, as in man, as the point of origin for the Ciliary Muscle, and only as such, and that none of its fibres bend into the Iris Root or take part in the formation of the Iris pillars. The way in which in lower animals the Iris is attached by a series of strands to the Cribriform Ligament gives the Angle a honeycombed appearance—an appearance which is responsible for the common assertion that the Iris pillars and

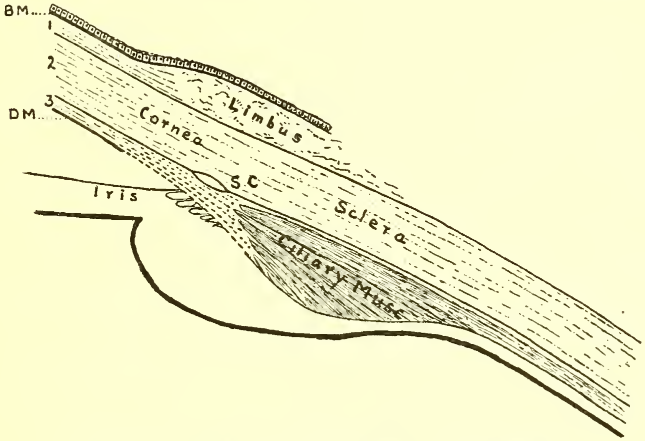

FIG. 17.—COMPARATIVE ANATOMY OF THE ANGLE.

As in man, the angle of the Anterior Chamber is formed by the Cribriform Ligament on the outer side, and the Iris Root on the inner. In most animals the Iris Root breaks up into a series of pillars, which cross the region of the Angle to become attached to the Ligament. The Apex of the Angle thus appears honeycombed.

The fibres of the Cribriform Ligament always act as the point of attachment for the fasciculi of the Ciliary Muscle, while the absence of the Scleral Ring makes the passage of aqueous into the Supra-Choroidal Space easier.

The Ciliary Processes invariably arise from a base which is continuous with the stroma of the Iris Root.

The peripheral termination of the superficial layers of the Cornea in the Limbus, and of the deeper layers in the Cribriform Ligament, make a natural anatomical division of the cornea into—(1) A Conjunctival Division, consisting of Epithelium, Bowman's Membrane (B.M.), and superficial corneal layers; (2) a Scleral Division; (3) a Uveal Division, comprising the inner Corneal Lamellæ, Descemet's Membrane (D.M.), and its Epithelium.

the fibres of the Cribriform Ligament are one and the same structure, which together constitute the "Ligamentum Pectinatum Iridis." Such an arrangement I have been unable to confirm in any of the animals examined in which the same uniform plan and analogous conformation exists as in man—*i.e.*, the angle is formed on the outer side by the Cribriform Ligament, and on the inner side by the Iris Root, which is attached to the Ligament. In man the apex is formed by the junction of the surfaces of these two structures, but in animals it is not such a definite point, but a region traversed by the strands composing the Iris pillars (Fig. 17).

In the lower animals the Iris is a more fibrous and much denser structure than in the human eye, and the crypts are proportionately less numerous; though it is important to note that, in consequence of the absence of the Scleral Ring and the presence of the pillars of the Iris, the route from the Anterior Chamber into the Supra-Choroidal Space is much more direct (Fig. 17).

As one would naturally expect, the conformation of the Angle in a monkey's eye closely approaches the human type, particularly in the fact that the Iris tissue consists of a loose stroma containing numerous crypts, and that its root does not break up into pillars, but is attached directly to the Cribriform Ligament.

The origin of the fibres of the Cribriform Ligament as a continuation of the Inner Lamellæ of the Cornea is easy to establish in animals, because of the pigmentation normally existing in and about this region, which delineates the individual corneal fibres of origin in a most striking manner.

I have also noted in all the animals examined the same peripheral termination of the superficial corneal fibres in the stroma of the Limbus as I have already described as existing in man.

Bowman's Membrane is variously developed in different animals, for in the pig it is absent as a distinct thick membrane; but the Limiting Membrane of the surface Epithelium spreads continuously underneath the corneal and conjunctival Epithelium, a point of some interest because in other animals, as well as in man, Bowman's Membrane is continuous with the Limiting Membrane of the Conjunctiva, a fact which strongly suggests the common origin of these two formations.

Summary of the Anatomical Features of the Angle of the Anterior Chamber.

1. The Angle of the Anterior Chamber is a true angle, formed by the attachment of the Iris Root to the Cribriform Ligament.

The apex is so situated that a line connecting the posterior extremity of Schlemm's Canal with the most anterior of the circular fibres of the Ciliary Muscle falls outside of it.

2. The appearance of the Angle as a sinus is an artificial condition produced by detachment of the Ciliary Body following fixation in Müller's Fluid.

3. The Cribriform Ligament is composed of a regularly-arranged lattice-work of circular and longitudinal fibres, which are a direct continuation of the similarly-directed fibres of the corneo-sclera.

Anteriorly the fibres are derived from the innermost lamellæ of the cornea, while posteriorly they divide into—

(*a*) A small external or scleral set, which becomes incorporated and lost in the sclera.

(*b*) A large internal or muscular division, which acts as a true ligament of origin to the Longitudinal fibres of the Ciliary Muscle, and as a check ligament to the Circular fibres.

4. The open network of the Cribriform Ligament allows the Aqueous direct access to Schlemm's Canal, and at the same time, in consequence of its origin, the Aqueous comes into contact with the inner lamellæ and interstitial spaces of the Cornea, while through its termination the Aqueous gains admission to the Supra-Choroidal Space.

5. The Cribriform Ligament is at birth a cellular formation, which undergoes progressive sclerosis with advancing years, till it ultimately becomes a fibrous structure.

6. Sclerosis creates an increasing mechanical obstruction, which predisposes to Glaucoma by impeding the access of aqueous both to Schlemm's Canal and to the venous channels traversing the Supra-Choroidal Space.

7. In the act of accommodation the circular bundles of the Ciliary Muscle pull the innermost fibres of the ligament taut, so that, instead of an anatomical deepening of the Angle, there is a physiological deepening produced by a facilitated access of Aqueous into the Supra-Choroidal Space.

The Ciliary Processes.

The position of the Ciliary processes in eyes fixed in Müller's fluid is very different from that in Formalin-hardened globes, owing to the artificial detachment of the Ciliary Body which follows fixation in the former fluid (Figs. 2 and 3). This difference must be borne in mind in considering the position of the Ciliary Processes in relation to the Iris Root.

The apex of the Angle of the Anterior Chamber in a Formalin-fixed globe is formed at the point of junction and attachment of the Iris Root to the Cribriform Ligament, and therefore the diameter of the chamber from Angle to Angle and the diameter of the Iris are the same.

In the adult eye this measurement varies within fairly large limits from 10 to 12 millimetres, thus approximately giving the chamber a perimetry of from 30 to 36 millimetres.

Now, the Ciliary Processes being about seventy in number, it follows that there are not more than two and a half, or less than two, Ciliary Processes to each millimetre of the Iris circumference. As the individual breadth of the Ciliary Processes is from 0·15 to 0·2 millimetre, it likewise follows that the area of the Iris circumference occupied by each Ciliary Process and its adjacent inter-Ciliary groove varies from 0·4 to 0·5 millimetre. This means that each Ciliary Process is separated from its neighbour by a space which is not less than its own breadth. The method of serial reconstruction is therefore an indispensable necessity if one desires to obtain a clear idea of the relation and connection of the Iris Root to the Ciliary Processes and their interspaces. The reconstruction of serial sections shows that the appearance of the attachment of the Iris Root to the Ciliary Body varies, depending on whether sections run through a Ciliary Process (Fig. 18) or through an Inter-Ciliary groove (Fig. 19). This difference is due to the interspaces or valleys between the Processes being deeper set than the bases from which the Processes themselves arise. Each Ciliary Process arises from a base which is a direct continuation of the stroma of the Iris Root (Fig. 18), while the valley or bottom of each Inter-Ciliary groove dips down so that the uveal pigment layers lie in almost direct contact with the mass of the Ciliary Muscle. The Inter-Ciliary spaces or

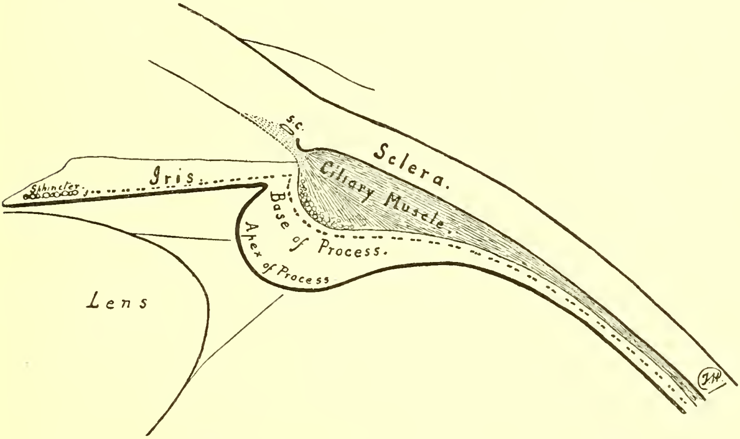

FIG. 18.—RELATION OF IRIS ROOT TO CILIARY PROCESSES.

Each Ciliary Process arises from a base which is continued forward into the stroma of the Iris Root. The dotted outline marks the bottom of the Inter-Ciliary groove and its continuation into the Iris Radial Furrow.

grooves are continued forward on to the back of the Iris as radial furrows (Fig. 19).

Fig. 18 is reconstructed from serial sections, and illustrates in heavy outline the connection and relationship of the Ciliary Body and Processes to the Iris Root, and in broken outline the continuation of the Inter-Ciliary groove with the Radial furrows of the Iris. Fig. 19 shows the converse. The difference between the base of the Processes and the bottom of the Inter-Ciliary groove can only be brought out by a process of serial reconstruction ; for as the Ciliary Processes are placed circumferentially, individual sections will only be able to show all and every kind of possible combination between the two extremes—between the full height of a Ciliary Process arising from its base (Fig. 18), and the bottom of the Inter-Ciliary grooves continuous with the Iris radial furrows (Fig. 19).

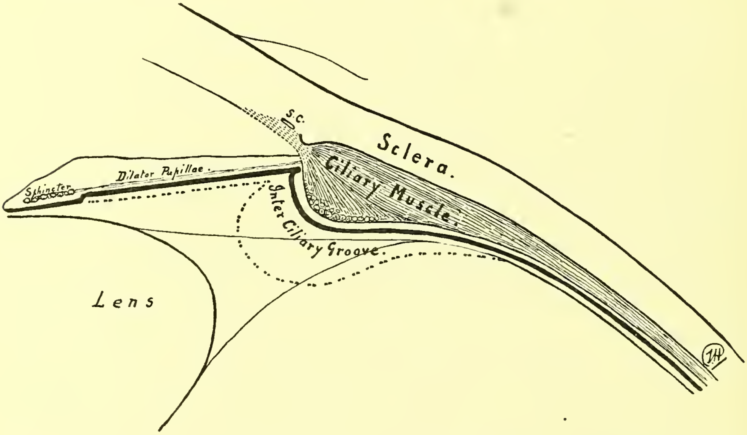

Fig. 19.—Relation of Iris Root to the Inter-Ciliary Grooves.

The pigment epithelium at the bottom of the Inter-Ciliary grooves comes almost in direct contact with the mass of the Ciliary Muscle.

Each interspace is continued on to the back of the Iris as a radial furrow, which stops abruptly at a point opposite the outer circumference of the *Sphincter Pupillæ*. The isolated fasciculi of the *Dilator Pupillæ* run immediately anterior to the Iris furrows.

The important points established by serial reconstruction are, first, that the Ciliary Processes arise from a base continuous anteriorly with the Iris Root, and, secondly, that the Inter-Ciliary grooves are prolonged into the Iris as Radial Furrows.

These anatomical details have a considerable pathological import, as they account for the position occupied by the Ciliary Processes when the Iris is displaced. The Iris Root cannot alter its position without causing traction on the Ciliary Bases, which in consequence will carry the superimposed Processes with them (*vide infra*, Fig. 42).

In sectioning an eye, one may strike a Ciliary Process, and at the same time include in the section the outer portion of a radial furrow of the Iris, and in this way produce an appearance as if the Ciliary Process had compressed the Iris Root, an appearance still further heightened in the case of globes fixed in Müller's Fluid by the rotation forward of the Ciliary Body (Figs. 20 and 44).

This "compression" of the Iris base, having been noted by Priestley Smith in cases of Glaucoma with open Angle, has been brought forward as evidence in supporting his theory. Serial sections show, however, that in Glaucomatous globes with open angle, as well as in normal eyes, this appearance has nothing to do with compression, but that it is purely the result of the normal anatomical relationship of the parts and the way they are sectioned, as it is only present in those sections which happen to strike a radial furrow of the Iris, and not in the adjoining sections of the series.

Fig. 20 shows such an apparent indentation of the Iris by the Ciliary Processes, but this "compression" is purely artificial, for, as seen in the adjoining serial sections, it is produced simply by the outer part of a radial furrow which this particular section has happened to strike. The way this section runs is shown by the dotted line in Fig. 21, which is a transverse section at the level of the apices of the Ciliary Processes from the same case. This transverse section shows three radial grooves, of which only the central one is cut truly transversely. The depth of this central

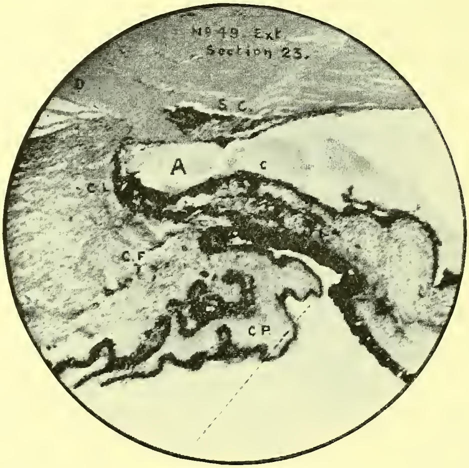

Fig. 20.—Micro-Photograph showing Apparent Compression of the Iris Root by the Ciliary Processes (C.P.) in a Normal Eye removed by Mr. Kingdom for Orbital Sarcoma, and in which I injected Indian Ink into the Anterior Chamber within Half an Hour after Enucleation.

This appearance is produced by this particular section striking a radial furrow of the iris pigment layers, and then coming out of it (*cp.* Fig. 42).

The injected ink has made its way into the iris stroma only where crypts (C.) are present.

This globe having been fixed in Müller's fluid, the ciliary body is detached (D.) and rotated forward round (*cp.* Fig. 3). The cribriform ligament is in consequence relaxed, and the angle of the anterior chamber appears as a rounded circular sinus (A.) instead of a true angle.

Compare the relationship of the parts here with that in a specimen fixed in formalin, as in Fig. 9, in which a line between the posterior end of Schlemm's canal (S.C.) and the circular fibres of the ciliary muscle (C.F.) falls altogether outside the angle. The dotted line represents position of transverse section (Fig. 21).

groove corresponds to the amount of the so-called "compression" of the Iris Root seen in the meridional section (Fig. 20).

In the rabbit the Ciliary Processes spring from the posterior aspect of the Iris Root, while in the dog, cat, and pig, they have slipped lower down, though they are still placed in front of the most prominent point of the belly of the Ciliary Muscle, so that their connection with the Iris Root is still very marked (Fig. 17). In the human eye this association between the two is not at all striking, and can, in fact, only be effectively made out by such serial reconstruction as shown in Figs. 18 and 19. The Ciliary Processes in man have moved still farther back, so that they come to lie on the top of, and extend behind, the most prominent part of the Ciliary Muscle; but their original association with the Iris is not altogether lost, as the ciliary bases are still continuous with the Iris stroma.

The Dilator Pupillæ.

An anatomical point, not directly concerned with the question of Glaucoma, and yet showing the great value of the study of serial sections, may be here referred to in connection with the Iris radial furrows, and the still vexed question of the nature and structure of the Dilator Pupillæ.

Of recent years* the view has been gaining ground that the Posterior Limiting Membrane of the Iris and the anterior of the two pigmented layers of the Pars Iridis Retinæ combine to form an ectodermic Dilator Pupillæ Muscle. The pigmented cells, according to this view, constitute the cellular elements of the Muscle, whose fibres lie packed together in the Posterior Limiting Membrane.

Serial sections disprove altogether the construction of a Dilator Muscle on such lines, by demonstrating, even in unbleached sections, the continuity of the Limiting Membrane of the Iris with the hyaline layer of the Ciliary Body, which layer in its turn is continuous with the Lamina Vitrea of the Choroid.

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* Fuchs's "Textbook," 3rd edit., p. 278.

These three formations are all one and the same continuous structure, acting as a Membrana Propria to the outer layer of cells of the secondary Optic Vesicle, and formed by their cellular activity in the same manner that Descemet's Membrane is laid down by the Posterior Corneal Endothelial Cells. Like Descemet's Membrane, this continuous membrane progressively thickens with age, so that although normally it is never so developed behind the Retinal pigment layer as it is over the Ciliary Body and Iris, yet in a senile eye it is throughout its extent considerably thicker than it is in a Child's eye. As the membrane is continued from the Ciliary Body into the Iris, it must perforce lie with a double curve, a circumferential as well as an antero-posterior one, so that a very slight obliquity in either of these two directions will obscure in the vast majority of sections the continuity of the two membranes.

Serial reconstruction of the Iris shows that the Dilator Pupillæ is not a continuous structure, for such would be quite unnecessarily powerful for its antagonistic sphincter; but that it consists of isolated radial fasciculi of non-striped muscle fibres arranged around and in front of the radial furrows, immediately anterior to the posterior limiting membrane of the Iris (Fig. 19).

The fasciculi of the Dilator Pupillæ have, as a point of origin, the innermost strands of the Cribriform Ligament, just before these latter fibres become lost and incorporated in the connective-tissue stroma of the Ciliary Muscle (Fig. 10, D.P.). From this origin the isolated fasciculi of the Dilator course radially inwards, to terminate by a direct insertion into the connective-tissue stroma of the Sphincter Pupillæ (Fig. 19).

After a study of serial sections, it is easy to understand the discrepancy which has arisen among histologists as to the presence or not of a Dilator Pupillæ Muscle. The Dilator Muscle is composed of non-striped Muscle fibres grouped in isolated fasciculi, which are only to be found lying in front of the Iris Radial furrows, and it is only in such sections as happen to strike the radial furrows that the muscular fibres are to be noted, as elsewhere no such formation can be made out.

Fuchs's Iris Crypts.

The anterior Iris surface is marked by elevations and depressions forming "the relief of the Iris." The pitlike depressions noticeable in the pupillary and ciliary areas of the Iris lead directly into the Iris stroma, and "place its tissue spaces in free communication with the cavity of the Anterior Chamber"* (Fig. 21).

The Crypts allow of the access of aqueous to the Iris Veins, and thus play the same part in relation to the Iris Veins as the Cribriform Ligament does in connection with the Venous Sinus of Schlemm's Canal; both crypts and perforated Ligament allow of the free and ready access of aqueous to Veins.

The endothelium covering the anterior Iris surface is described as stopping at the openings of the crypts, but serial sections show no break, but complete continuity, between the flattened laminated surface cells of the Iris and those composing its stroma.

At the mouths of the crypts every variety and transitional form of cell can be met with between typical endothelial and equally typical alveolar cells. The one variety of cell shades off into the other. At the crypts we have another example of the unity and anatomical continuity of "endothelial" and "connective - tissue" cells, such as has already been illustrated as existing in the case of the endothelial cells of Descemet's Membrane and the covering cells of the fibres of the Cribriform Ligament, which in their turn are associated with the corneal interspaces and corpuscles.

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* Fuchs's "Textbook," 3rd edit., p. 278.

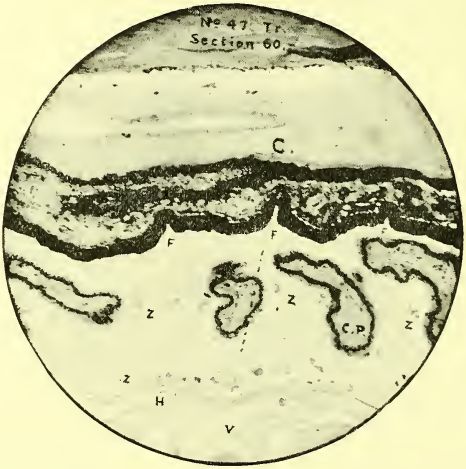

Fig. 21.—Micro-Photograph of Transverse Section of Iris at the Level of the Apices of the Ciliary Processes, from the Same Case as Fig. 20, showing Furrows of the Iris Pigment Layers (F. and F.′).

The two lateral folds (F.′) are not sectioned quite truly transversely, as must naturally happen, considering that these folds run radially. Iris crypt (C.) filled with ink. Apices of ciliary processes (C.P.). In the interciliary spaces lie the transversely-cut zonular fibres (Z.), which also form a line in front of the hyaloid membrane (H.) of the vitreous (V.).

The dotted line represents the direction of a meridional section that would give rise to an appearance of apparent compression of the iris root if the section proceeding centrally were suddenly to leave the furrow, as has happened in Fig. 20.

In the further sections of this series the radial furrows (F. and F.′) can be traced as direct continuations of the inter-ciliary grooves (*cp.* Fig. 19).

The depth of the transversely-cut radial furrow (F.) corresponds to the amount of the "compression" seen in Fig. 20.

The term "endothelium" is a convenient one, first introduced by His, to designate an epithelium-like tissue of mesodermic origin; but if the expression be pushed too far it creates an artificial separation between cells of the same elementary tissue. This objection is specially applicable to the case of the cells lining the Anterior Chamber.

The flattened cells forming the so-called anterior endothelium of the Iris are only the superficial layer of cells of the Anterior Limiting Membrane, which latter formation is but a condensed stratum of the connective tissue of the Iris stroma.

The Iris is not the only tissue in the Body in which the connective tissue on its superficial aspect takes on an epithelioid appearance, for, in describing the flattened or lamellar cells of connective tissue in general, Schäfer* says: "The cells in some parts are united by their edges into patches, after the manner of an epithelium; in other cases, a union takes place by means of branched processes, so that the cells form a kind of network throughout the ground substance. In many membranous forms of connective tissue" (*inter alia*, the Iris), "the flattened cells form an epithelial-like covering to the surface of the membrane, and may even complete the latter by bridging over any gaps existing between the bundles of fibres forming the membrane. Such epithelioid tracts may be of considerable extent. It is often observable that the cells at the margin of the patch have processes at their border, which are connected with the ordinary scattered cells of the tissue."

This description of the general connective tissue of the body is applicable in every way to the Iris stroma as a whole, and to its anterior aspect in particular. The anterior limiting membrane of the Iris is composed of a condensed layer of cells of the connective-tissue stroma, and the so-called "endothelial layer" is nothing more than its superficial layer of flattened-out connective-tissue cells arranged close together, edge to edge. In this manner are formed areas in which, it is true, patches of cells present an epithelioid appearance, but the cells in question exhibit every transition to the other cells of the connective-tissue stroma, a fact which is most evident where they bridge over spaces and holes, and where they approach the openings of the Iris crypts of Fuchs. The posterior corneal endothelium is responsible for the formation of Descemet's Membrane, a structure laid down by a process of secretion or deposition; hence its increasing thickness the older the eye. If the superficial layer of cells on the anterior Iris surface were a direct continuation of the posterior corneal endothelium, one would naturally expect them to lay down a homogeneous membrane analogous to Descemet's Membrane. Such a homogeneous membrane is never found in the normal Iris. Pathologically—as, for instance, in Glaucoma—after the Iris tissue has once become atrophied, the dominating activity of the posterior corneal endothelium asserts itself, so that it spreads over the Iris surface, and in its progress lays down a homogeneous membrane. This will not, however, occur without Glaucoma and subsequent Iris atrophy, for in ordinary traumatic or post-operatic synechiæ, even of old standing, the corneal endothelium never spreads over the Iris surface.

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* Quain's "Anatomy," 10th edit., p. 231.

But, further, while the posterior corneal endothelium is very prompt in covering over defects in its continuity, so that the inner aspect of corneal incisions is readily sealed by a growth of endothelium, wounds in the healthy Iris never manifest any reparative changes (*vide infra*), and the severed surfaces do not, even years after, become covered by the endothelial layer, which would certainly be the case if this layer were a continuation of, and of the same nature as, the posterior corneal endothelium. It is therefore necessary on morphological, physiological, and pathological grounds to make a distinction between the posterior corneal endothelium and the lining cells of the Anterior Iris surface.

The two respective layers of cells have, however, this one function in common, that they prevent the direct passage of Aqueous into the tissue which they cover. The corneal endothelium prevents the Aqueous passing directly into the Corneal tissue, which it can only do indirectly through the origin of the Cribriform Ligament; while, similarly, the Aqueous cannot pass into the Iris Stroma except by way of the Iris crypts. This fact is well brought out in Figs. 20 and 21, which show the normal Iris after injection of Indian Ink into the Anterior Chamber. The pigment has made its way into the Iris Stroma only through the open mouths of the Crypts, and has then spread along the posterior aspect of the Iris stroma, leaving the anterior half devoid of pigment; this would not be possible if the pigment had been able to pass directly through the anterior Iris surface. Thus, the Iris surface, it is important to emphasize, is not a resorbing area in the ordinary sense of the word, but it acts as such solely because of its crypts, which allow the Aqueous direct passage to the Iris Veins.

In animals the Iris is a more fibrous structure, and crypts are either poorly developed or absent altogether, and therefore the amount of Indian Ink in the Iris after injection into the Anterior Chamber is proportionately reduced.

In the human eye, however, the loose Iris stroma and the numerous and well-marked crypts present in their function a close analogy to the Cribriform Ligament, as both open crypts and perforated ligament allow the Aqueous to come into direct contact with Intra-ocular veins.

Summary of the Main Anatomical Features of the Ciliary Body and Iris.

1. The Ciliary Processes arise from bases which are continuous with the stroma of the Iris Root.

2. The Inter-Ciliary grooves are prolonged on to the posterior surface of the Iris as Radial Furrows.

3. The Posterior Limiting Membrane of the Iris and the Hyaline Layer of Ciliary Body and Choroid form one continuous sheet.

4. The Dilator Pupillæ Muscle consists of isolated fasciculi of non-striped muscle fibres, which are only to be found immediately in front of, and parallel with, the Iris Radial Furrows.

5. The Iris Crypts lead the Aqueous direct to the Iris Veins.

Schlemm's Canal and the Anterior Uveal Veins.

The structure of Schlemm's Canal, and its immediate relationship to the Cribriform Ligament on the inner side, and to the sclera on the outer, are best made out in serial sections of senile eyes with total sclerosis of the Ligament. In such eyes a sharp contrast exists between the single layer of the endothelial cells, which, resting on a basement membrane, compose the sinus wall, and the now sclerosed fibres of the Cribriform Ligament.

The statement that the walls of Schlemm's Canal are firmly adherent to the surrounding sclerotic, so that the lumen is kept constantly open,* is quite erroneous, as it lies perfectly free in its niche, attached by nothing more than its tributary vessels, so that between the sinus and the scleral fibres there exists a potential space which may be injected with Indian Ink (Fig. 22). The existence of this space may also be clearly shown in cases of Keratitis Punctata as well as in lower animals, in whom pigmentation in and about the Cribriform Ligament is a normal condition.

In form Schlemm's Canal is an irregular ellipse with a long diameter of about 0·3 millimetre, and a short one of about 0·05 millimetre; while in consequence of its position it lies parallel with the innermost fibres of the Cribriform Ligament, so that its Anterior extremity is tilted somewhat inwards, and its posterior end somewhat outwards (Figs. 9 and 10).

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* Parsons, *op. cit.*, p. 986.

Its lumen often appears subdivided into two, three, or more divisions, owing to the fact that while encircling the Angle of the Anterior Chamber it receives afferent and gives off efferent vessels, so that at the junction of these tributaries individual sections will show the sinus as if split up into a number of partitions (Fig. 22).

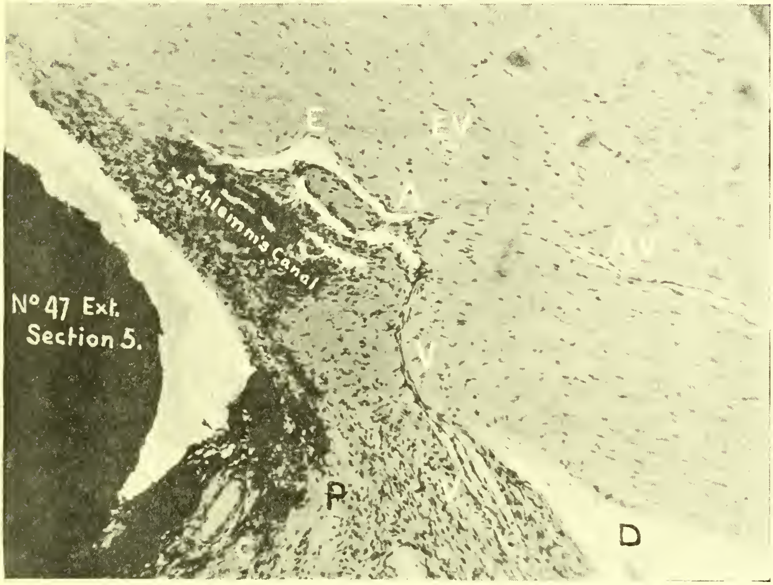


Fig. 22.—Micro-Photograph of Schlemm's Canal, also from the Same Case as Fig. 20.

Schlemm's Canal appears broken up into a number of divisions, owing to its formation by a plexus of vessels. Into this plexus runs a vein (V) from the Ciliary Muscle venous plexus, and at A another afferent tributary (A.V.)

The Circulus Iridis Major (C) is surrounded by pigment (P) which has impregnated the heads of origin of the fasciculi of Ciliary Muscle. The network of the Cribriform Ligament is filled with pigment which has also passed here internal to Schlemm's Canal. Efferent vein (E.V).

The now well-recognized fact, first and foremost insisted on by Leber, that Schlemm's Canal is a venous sinus, does not by itself assist us to comprehend why this particular venous system should play the preponderant part assigned to it in the physiology of aqueous resorption, considering that, as discussed in the preceding section, the open mouths of Fuchs's Iris Crypts allow of the ready access of the Aqueous to the Iris veins, and thus these latter should act as equally active agents as Schlemm's Canal itself. The answer to such a question is to be found in the anatomical connections of the sinus.

Considering the controversy that has raged concerning the nature of Schlemm's Canal, and the amount of experimental work that has been undertaken in support of the view of a lymphatic as opposed to a venous nature of the sinus, it is strange that the idea of approaching the question from what is, after all, the only proper quarter—the anatomical—has not before now been thought of, and the connections of Schlemm's Canal traced out by reconstructing serial sections.

In serial sections it is perfectly easy to follow from section to section the course and connection of every vessel, and to trace all branches to their termination. Individually and at random such sections do not show anything that could in any way seriously modify Leber's conclusions, but studied collectively and seriatim they demonstrate connections altogether inconsistent with accepted teaching.

The vascular supply of the anterior portion of the Uvea is derived, according to Leber, whose teaching is followed in all textbooks, from the Anterior Ciliary Arteries, reinforced by the Posterior Ciliaries, which jointly form the Circulus Arteriosus Iridis Major. This arterial circle supplies the Ciliary Body and Iris, in which latter structure its branches form the Circulus Iridis Major.

In respect to this accepted arrangement of the intraocular vascular supply, one may well ask what purpose is subserved by this extraordinarily large arterial supply to the

Iris, which is quite out of proportion to its anatomical structure and known physiological demands; for nowhere else in the human structure does the arterial supply to a part so obviously exceed its requirements as is apparently the case in the Iris.

The whole of the venous blood within the eye is described as being returned by the Venæ Vorticosæ, with the exception of that from a small area on the outer side of the Ciliary Muscle which is drained by the small anterior ciliary veins. This description of the vascular arrangement within the eye is universally accepted, and is apparently perfectly correct if only a limited number of sections are examined; but

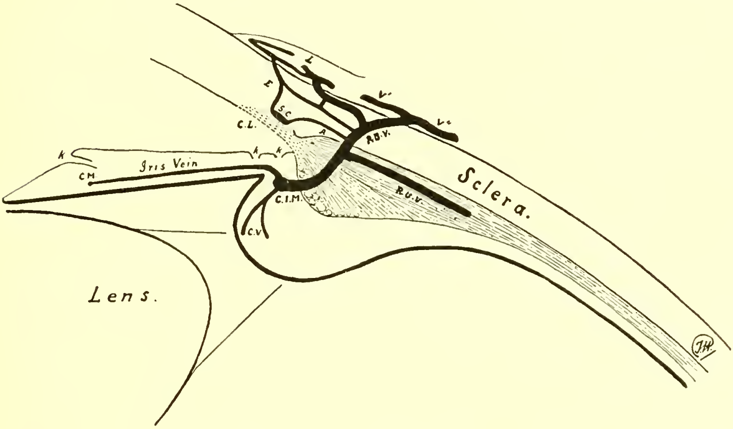


Fig. 23.—The Iris Venous Return.

The Circulus Iridis Major (C.I.M.) is formed by the Iris Veins through also receiving tributaries from Ciliary Veins (C.V.). Before piercing the Sclera, it gives off a branch to the Posterior Uveal Venous system (P.U.V.), and then it pierces the Sclera (A.U.V.) to take part in the Anterior Uveal Venous system (see Fig. 24). The main trunk supplies afferent tributaries (A) to Schlemm's Canal, as well as other branches which join the superficial plexus (L) in the limbus.

On the surface the trunk invariably divides into Anterior and Posterior branches (V′ and V″). The efferent vessel (E) from Schlemm's Canal (S.C.) is closely associated with the superficial pericorneal plexus (L). The Cribriform Ligament (C.L.) and Iris Crypts (K) lead the aqueous respectively to Schlemm's Canal and Iris Veins.

when an extensive and consecutive series of sections is studied, and a composite diagram made of the course of the vessels so traced, a picture is produced which differs fundamentally from Leber's well-known diagram, as reproduced in most textbooks.

According to Leber, there are two Anterior Ciliary Arteries derived from the arterial supply to each Rectus, and a similar number of Anterior Ciliary Veins, with which Schlemm's Canal is associated, so that the total number of vessels which perforate the Sclera at the pericorneal circumference only amounts to sixteen.

Serial sections show that Schlemm's Canal has far more extensive venous connections than have been ascribed to it, as all the vessels which perforate the sclera at the pericorneal circumference anastomose with each other, as well as being connected directly or indirectly with Schlemm's Canal. In spite of the most careful search by serial reconstruction, including one series which comprises more than half the circumference of the Anterior Chamber, I have failed to find a single vessel about the corneal circumference which was not thus connected. It follows that if all the vessels which perforate the sclera at the pericorneal circumference are connected with each other, and with Schlemm's Canal, they must all be veins, even the so-called Anterior Ciliary Arteries.

It is impossible to imagine that there can ever normally exist such direct connection between a so-called arterial trunk and the venous sinus of Schlemm's Canal, such as shown in Fig. 23, without disastrous results to the intraocular circulation and pressure, and therefore the only conclusion that one can come to is that these vessels are not arteries which supply blood to the Iris, but that they must be veins which return blood from the Iris.

But further serial sections show that what are recognized as anterior ciliary veins are much more numerous than they are stated to be, and that they have a very much deeper and more extensive origin than the mere outer side of the Ciliary Muscle, as they are connected with the capillary venous plexus existing in the interstitial tissue of the whole of the Ciliary Muscle as well as with the veins from the Ciliary Processes themselves (Fig. 24). The tributary

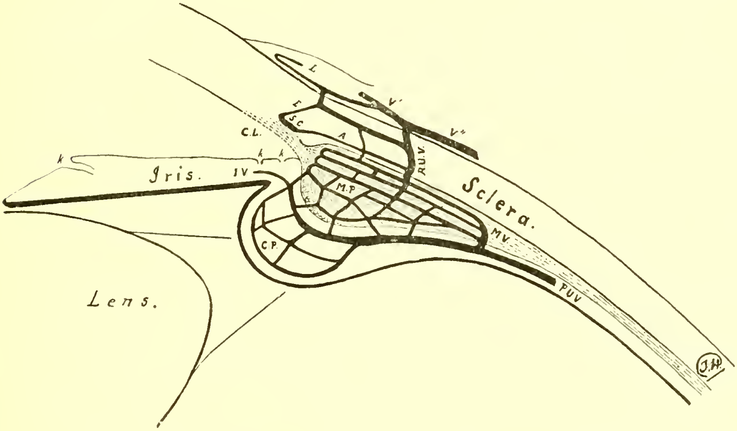


FIG. 24.—THE CILIARY VENOUS RETURN.

The Ciliary Plexus (C.P.) is closely associated with Iris Veins (I.V.) and with the Muscular Plexus (M.P. and M.V.) in forming branches (P.U.V.) which join the Posterior Uveal Veins or Venæ Vorticosæ. Anteriorly piercing the Sclera about the pericorneal circumference are the Anterior Uveal Veins, which derive their blood directly from the whole of the Ciliary Muscle Plexus (M.P. and M.V.).

The main vessels, as they pass through the sclera, all communicate with Schlemm's Canal either directly or indirectly, and on the surface break up into Anterior and Posterior Branches (V′ and V″). The former join the superficial pericorneal plexus (L) of the Limbus. The Ciliary Venous Return in its passage through the Sclera is thus closely linked with the Iris Venous Return (see Fig. 23) to form an Anterior Uveal Venous System.

vessels of Schlemm's Canal are thus derived, not merely from the outer portion of the Ciliary muscle, but from the venous system of the whole Ciliary Body and Processes, as well as from the Iris Veins; in short, from the venous return from the whole anterior portion of the Uveal Tract.

As the perforating scleral vessels about the pericorneal circumference drain the anterior portion of the Uvea, they may collectively be described as Anterior Uveal Veins, in contradistinction to the posterior uveal system of the Venæ Vorticosæ.

The Anterior Uveal Veins are derived from and drain two distinct regions of the Uvea—the Iris and the Ciliary Body. The veins from these two areas before perforating the sclera anastomose freely and directly with the Radicles of the Venæ Vorticosæ (Figs. 23 and 24), while at the same time in their course through the sclera they are linked together by a great number of intercommunicating branches, as well as by other and similar vessels which do not themselves completely perforate the sclera.

Each Anterior Uveal Vein, whether originally derived from the venous system of the Iris or Ciliary Body, besides its intercommunications with adjoining vessels, is always connected directly or indirectly by one or more branches with Schlemm's Canal. I have not yet found a single Anterior Uveal Vein which, traced in serial sections, did not supply either directly or indirectly afferent vessels to Schlemm's Canal, and receive afferent tributaries from that Venous sinus.

The total number of Anterior Uveal Veins is considerable, for in serial sectioning I have invariably found one perforating vessel to each millimetre of the inner corneo-scleral circumference, frequently two, and occasionally three. Taking the inner corneo-scleral perimetry at 30 millimetres, the total number of perforating veins lies somewhere between forty and fifty, possibly nearer the higher than the lower number, exclusive, too, of the large number of small non-perforating vessels. In consequence of the large number of the Anterior Veins and their free anastomosis in the Sclera, its pericorneal circumference is simply honeycombed by these venous channels and their intercommunication, so that the weakness of the Sclera in this area must be ascribed to these vessels, and not merely to the presence of Schlemm's Canal.

The Posterior Veins, or Venæ Vorticosæ, do not comprise the sole or even the main venous return from the Iris and Ciliary Body, as the prime duty of draining these areas devolves on the Anterior Uveal Veins. These vessels before perforating the Sclera communicate freely and directly with the radicles of the Venæ Vorticosæ, after which, on entering the Sclera, they are joined together by a large number of anastomosing branches, including the loops sent to and received from Schlemm's Canal.

In consequence of this close association between the two main Uveal Venous systems, it follows that the intravenous pressure in one part of the Uveal tract cannot be altered without tending to effect the venous pressure within the sinus of Schlemm's Canal.

The importance of Schlemm's Canal, as the chief physiological outlet of the aqueous, is due to its formation by tributary vessels derived from both Iris and Ciliary veins. It therefore follows that the blood-pressure in the afferent veins of Schlemm's Canal, and therefore of Schlemm's Canal itself, must be less—just slightly less—than that in the original capillary venous areas of the Iris and Ciliary Body. It is because the blood-pressure within it is the lowest with which the aqueous comes into direct contact that Schlemm's Canal owes its preponderant rôle in aqueous resorption.

But for the advantage that Schlemm's Canal has of having a slightly lower blood-pressure than that of the Iris Veins, there would be no anatomical reason whatsoever why the Iris under physiological conditions should not play an equally great part in resorption of aqueous as Schlemm's Canal itself; for, in consequence of the open mouths of the iris crypts, the aqueous is brought into direct contact with the walls of the iris veins, in precisely the same way as the Cribriform Ligament allows the aqueous to come into contact with the sinus walls of Schlemm's Canal.

As this close association between Schlemm's Canal and the Anterior Uveal Veins is of the highest importance in the physiology and pathology of the intra-ocular pressure, it is essential to discuss these connections in some considerable further detail.

The Venous Return from the Iris.

The Anterior Uveal Veins derived from the Iris are in number very much fewer than those that drain the Ciliary Body, though individually they are as a rule much larger vessels. The exact ratio of the one set of veins to the other I am not at present in a position to state, because of the extreme irregularity of the perforating Scleral branches which the Circulus Iridis Major gives off (*vide infra*, p. 70).

The connections of the Circulus Iridis Major with Schlemm's Canal constitute this Vascular Circle a true Sinus Venosus Iridis, in contradistinction to the Sinus Venosus Scleræ, or Schlemm's Canal. The direct connection between these two circles is well shown in Figs. 25 to 27, and again in Figs. 28 to 30. In the former series I am fortunate enough to be able to demonstrate the direct connections in a couple of adjacent sections, while in the latter series it has required three, which by themselves would be unconvincing but for the fact that the microphotographs represent Sections 19, 24, and 28, of the series. In other series it has required a considerable greater number of sections to trace out this association. Figs. 31, 32, and 33, show the perfectly straightforward though somewhat tedious process by which the connections are to be made out.

Figs. 31 and 32, illustrating different forms of connections between the Circulus Venosus Iridis and Schlemm's Canal, are the result of piecing together the first two hundred sections of a series which comprise more than half of a normal eye.

Fig. 33 represents a similar composite diagram, also from a normal eye, which has taken eighty sections to complete. In these composite pictures the numbers represented within circles correspond to the number of the section, and thus, while giving a sort of relief to the diagram, they show the absolute worthlessness of anything short of the complete series of sections to demonstrate the actual condition of affairs.

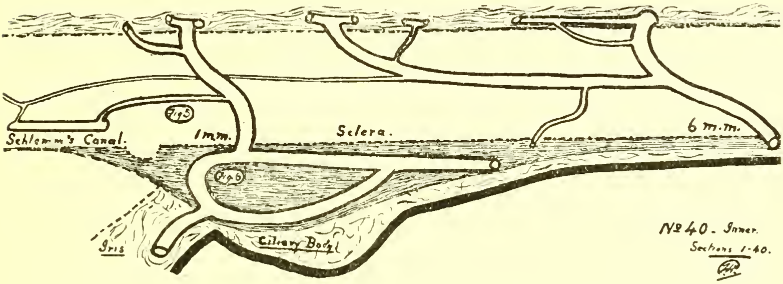


FIG. 25.—COMPOSITE DIAGRAM FROM SERIAL SECTIONS, ILLUSTRATING CONNECTION BETWEEN IRIS VEINS, CIRCULUS VENOSUS IRIDIS (SO-CALLED "CIRCULUS ARTERIOSUS"), AND SCHLEMM'S CANAL.

The posterior of the two perforating scleral vessels is an Anterior Uveal Vein derived from the choroid. This vessel is also connected with Schlemm's canal. Figs. 5 and 6 refer to micro-photographs shown in Figs. 26 and 27.

The connections shown in these composite diagrams are by no means the only ones, but they are the most direct and obvious links between the Circulus Iridis Major and Schlemm's Canal. All the other numerous vessels and branches which the sections showed have been left out.

In Fig. 31 is traced a branch which is given off by the

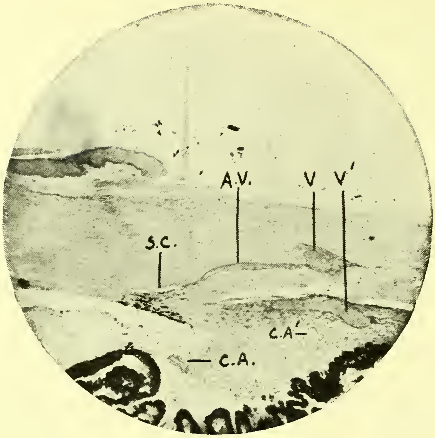


Fig. 26.—Micro-Photograph of Section belonging to the Series from which the Diagram (Fig. 25) was reconstructed.

Schlemm's canal (S.C.) receives at its posterior margin an afferent vein (A.V.) from the perforating scleral vessel (V), which is a continuation of V′ and of the so-called "circulus arteriosus" (C.A.), as shown in Fig. 27.

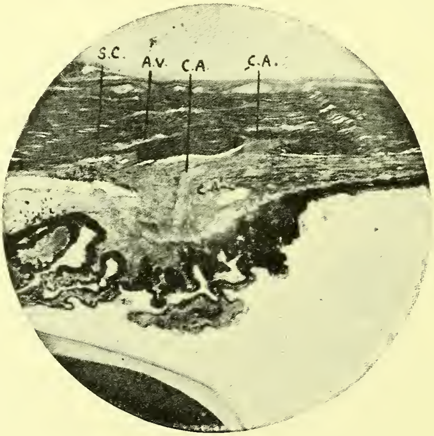
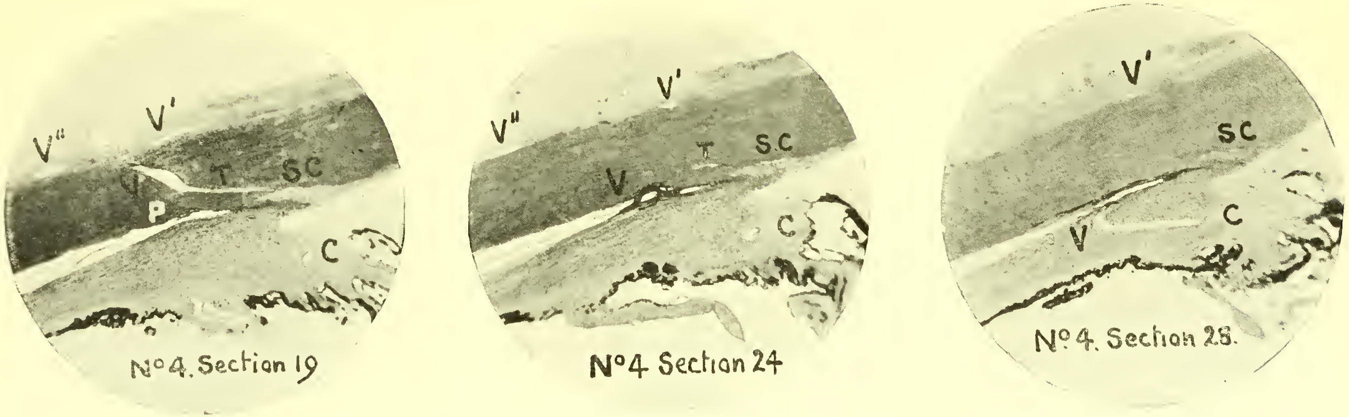


Fig. 27.—Micro-Photograph of a Section Adjacent to that illustrated in Fig. 26, which completes the Connection existing between this so-called "Anterior Ciliary Artery" (C.A.) and the Perforating Scleral Vessel (V and V′) of Fig. 26.

Schlemm's canal (S.C.) with now only a small portion of afferent vein (A.V.).



FIGS. 28, 29, 30.—MICRO-PHOTOGRAPHS FROM SERIAL SECTIONS ILLUSTRATING THE VENOUS NATURE OF THE CIRCULUS IRIDIS MAJOR.

Fig. 28 (Section 19) : Schlemm's Canal (S.C.) receives a tributary from the Vein (V), which latter breaks up on the scleral surface into two branches (V' and V''). The Circulus Iridis Major (C) is seen in transverse section.

Fig. 29 (Section 24) : The Vein of Section 19 here seen crossing the Supra-Choroidal Space, and surrounded by a mantle of pigment. The Circulus Iridis Major (C) has moved outwards.

Fig. 30 (Section 28) : Here is shown the continuity as one and the same vessel of the Vein and the Circulus Iridis Major.

Circulus Venosus Iridis, and which connects it with the Anterior Uveal system of veins and Schlemm's Canal.

It is irregular branches such as this which make it difficult to estimate the proportion of Iris to Ciliary Veins which enter into the formation of the Anterior Uveal System of Veins. The connection between the Circulus Iridis Major and Schlemm's Canal, which the first one hundred sections of this series show, is perfectly direct and straightforward.

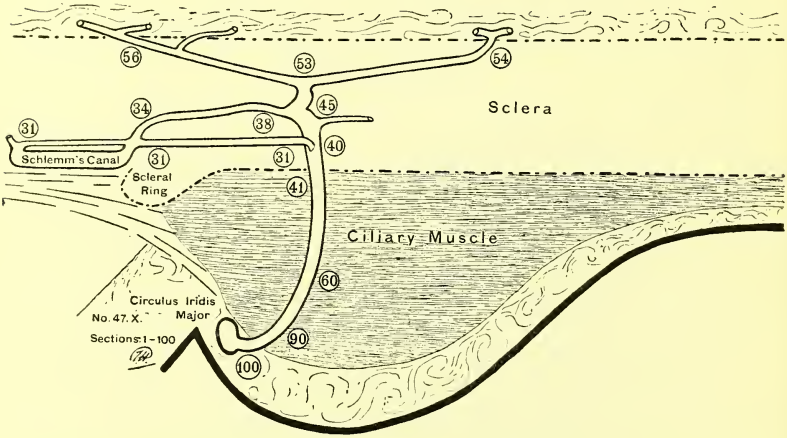


Fig. 31.—Reconstruction of Connections of Circulus Iridis Major.

The figures in circles denote the number of the sections which made up the series. Section 31 shows in longitudinal section a tributary of Schlemm's Canal, which is connected with a vessel which, in its turn, is a branch of the Circulus Iridis Major.

Section 31 shows in longitudinal section a tributary of Schlemm's Canal, whose distal end is cut transversely in Sections 32 to 39. In Section 40 this tributary is seen connected with an Anterior Uveal Vein, which in the next section (41) is seen crossing the Supra-Choroidal Space. From then onwards this Anterior Uveal Vein is seen in transverse section, moving gradually inwards, till in Section 100 it joins the Circulus Iridis Major. Thus throughout seventy sections one can trace with absolute ease this branch from the Circulus Iridis Major moving slowly outwards to take part in the Anterior Uveal Venous System, into which this particular vessel breaks up and anastomoses, though only a few of its main divisions are here shown.

Fig. 32 shows the continuation of the series of Fig. 31. The Circulus Iridis Major moves gradually outwards and backwards into the Ciliary Muscle, and at Section 80 it divides into an Anterior and a Posterior Uveal Division. The Anterior Uveal Branch in its passage through the sclera divides (in Section 160) into two subdivisions, which pass respectively forwards and backwards. The fact that the division takes place in the sclera, and that both vessels emerge on the surface, is further indication of the course of the blood-stream, as flowing from within out-

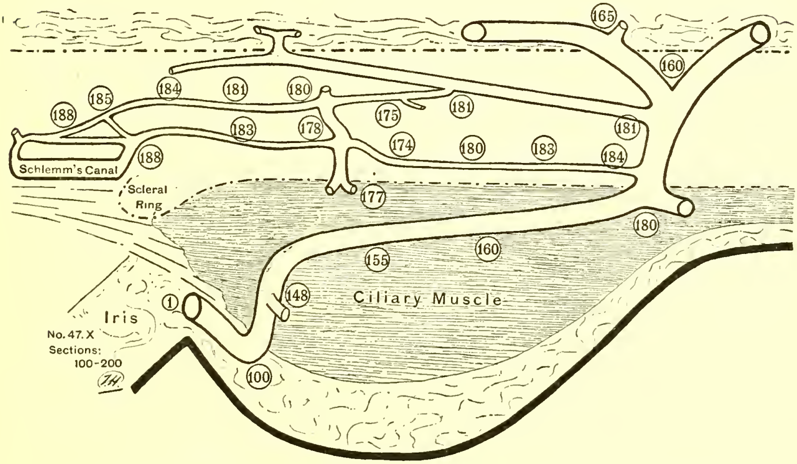


Fig. 32.—Further Connections of Circulus Iridis Major from the Next Hundred Sections from the Same Case as Fig. 31.

In Sections 1 to 100 the Circulus Iridis Major is seen in transverse section, after which it moves outwards, and perforates the Sclera in Section 160 in two main branches—an anterior and a posterior. In its course through the Sclera the vessel is linked with Schlemm's Canal by an Anterior Ciliary Vein.

wards, since one could hardly expect an arterial vessel to divide in such a position and in such a manner against what would be the current of the arterial flow.

The illustrations so far shown have been examples of direct connections between the continuation of the Circulus Iridis Major and Schlemm's Canal, but here we have an example of an indirect association brought about by a small vessel which does not itself completely perforate the Sclera. This vessel, formed by the junction of several small muscular twigs, after crossing the Supra-Choroidal Space, sends out two branches which form direct connecting links between the large perforating Anterior Uveal Vein and Schlemm's Canal. This same vessel is seen in Section 179 sending out two other branches, which form another link between Schlemm's Canal and the Iris venous return.

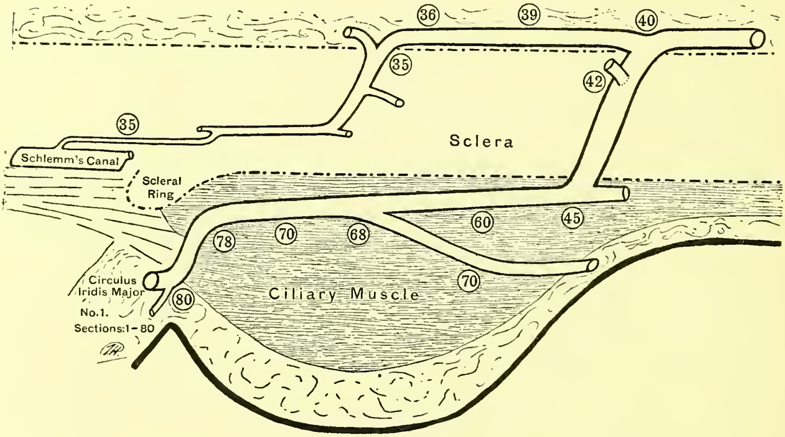


Fig. 33.—Reconstruction of Main Branches of Circulus Iridis Major, which between Sections 80 and 40 can be traced making its Way outwards, and, after perforating the Sclera, dividing on the Surface into an Anterior and Posterior Division.

The Anterior division is connected with Schlemm's Canal.

The composite diagram of Fig. 33 illustrates another form of indirect connection between Schlemm's Canal and the Iris Venous system. In this series Section 35 shows in longitudinal section the whole length of a tributary from Schlemm's Canal to the Scleral surface. Sections 36 to 40 trace the connection with an Anterior Uveal Vein, which is seen to perforate the Sclera in Sections 40 to 45. The next thirty-five sections trace the continuation of the Anterior Uveal Vein with the Circulus Iridis Major.

In Section 42 the Anterior Uveal Vein, while still in the sclera, gives off a branch which comes forward on the surface, and then runs parallel with the one shown in the illustration, with which it anastomoses, besides being similarly connected with Schlemm's Canal.

I have traced out these connections between the Circulus Iridis and Schlemm's Canal in literally hundreds of sections in normal as well as pathological eyes, with exactly the same result in all cases. In a Glaucomatous eye the picture produced is even more striking, owing to the formation of new channels producing a much closer vascular network, which simplifies the tracing of the association between the two venous circles of the Iris and Sclera.

The anastomoses between the Iris Venous return and Schlemm's Canal naturally divide themselves into three types or forms. The connection may, firstly, be direct from the Circulus Venosus Iridis; secondly, it may take place as the Iris Vein perforates the sclera; and, finally, it may occur on the surface of the sclera, where branches from Schlemm's Canal join branches from the Iris Veins. I have illustrated all these classes of anastomoses. The direct connection between the Circulus Venosus Iridis and Schlemm's Canal is shown in the composite diagram, Fig. 31. The direct tributaries to Schlemm's Canal given by the Iris Vein as it perforates the sclera are seen in the series of micro-photographs, Figs. 26 to 30, while the similarly-placed indirect anastomoses are seen in the reconstructed diagram, Fig. 32.

Finally, the connection between Schlemm's Canal and the surface branches of the Iris Veins is shown in Fig. 23 as well as in Fig. 33.

In conclusion one can state that, in view of the numerous direct and indirect communications shown to exist between Schlemm's Canal and the so-called Anterior Ciliary Arteries, it is quite impossible to imagine that these vessels can be anything else but veins, and not arteries.

Besides this free anastomosis with Schlemm's Canal, there is another factor which points to the venous nature of the Circulus Iridis, and that is the way in which its main vessels divide when still in the Sclera into Anterior and Posterior branches.

If these vessels were arterial trunks which carried the blood from without inwards, it would be against all reason for such branches to be given off in such a position and in such a manner as shown in the reconstructed diagrams, Figs. 23, 32, and 33, as well as in the micro-photograph, Fig. 28. Such a mode of branching directly against the supposed arterial blood-stream is certainly inconsistent with an arterial inflow, though perfectly compatible with a venous flow from within outwards.

The Venous Return from the Ciliary Body.

The Ciliary branches of the Anterior Uveal system of veins considerably outnumber the Iris branches, though individually the vessels are as a rule considerably smaller. The origin of these Ciliary Vessels is not merely the outer portion of the Ciliary Muscle, but embraces the whole of the capillary venous ramifications, permeating the interstitial tissue of the Ciliary Muscle, so that their origin is closely linked with that of branches the which run to the Venæ Vorticosæ (Figs. 24 and 34).

These deep connections of the Anterior Uveal Veins are well made out in serial sections from senile eyes stained after Van Gieson's method, which makes the vessel walls stand well out in sharp contrast to the sclerosed interstitial tissue of the Ciliary Muscle.

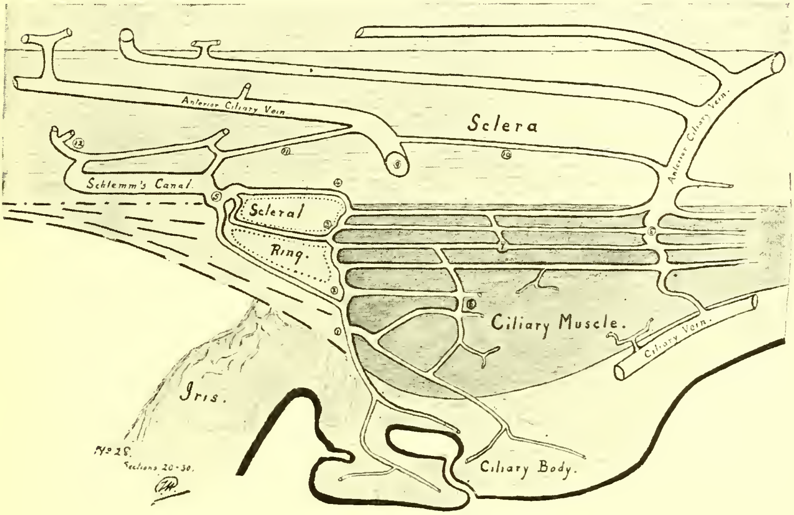


Fig. 34.—Reconstruction from Ten Serial Sections of Anterior Uveal Veins and their Connection with Schlemm's Canal.

(1) Veins from ciliary process, meeting to form a branch which runs directly outwards between, and anterior to, the fasciculi of the ciliary muscle. These vessels give off anteriorly tributaries to Schlemm's canal, and posteriorly branches which run in the interstitial tissue of the ciliary muscle. The direct afferent vessels to Schlemm's canal run internal to (2), through (3), and external to (4) the scleral ring, and they join Schlemm's canal at its posterior margin (5). In the interstitial tissue of the ciliary muscle lies a network of veins (6 and 7) which run together (8) to form an anterior uveal vein.

The anterior of the two perforating veins seen here (9) arises in exactly the same way—by deep branches from the ciliary process—as the posterior one with which it is connected (10). Afferent (11) and efferent (12) vessels of Schlemm's canal.

The Ciliary Body and Processes are permeated throughout by a capillary venous ramification, which links together the origin of the Anterior with the Posterior Uveal Systems. In the Ciliary Muscle the general course of the capillaries is always parallel with the fasciculi of the Muscle, there being at the same time free anastomosis among themselves as well as intercommunication with the capillary venous plexus of the Ciliary Processes. The vascular network so formed is a very close one, but its general plan is well illustrated in Figs. 24 and 34. A constant feature of the Ciliary Muscle Plexus is a vessel which runs directly outwards between, and anterior to, the heads of origin of the muscular fasciculi of the Ciliary Muscle. This vessel—Fig. 34 (1)—receives numerous tributaries from the venous capillaries of the Ciliary Process, and as it proceeds outwards is connected on the one hand with Schlemm's Canal, and on the other with the network of vessels coursing in the interstitial tissue stroma of the Ciliary Muscle. The afferent tributaries to Schlemm's Canal run internal to (2), through (3), and external to (4) the Scleral Ring. In the micro-photograph Fig. 10 is seen the junction (at ×) of an afferent vessel to Schlemm's Canal, which runs a similar course to that shown in Fig. 34 by the vessel marked 2. The micro-photograph Fig. 22 illustrates the whole length of another direct afferent tributary to Schlemm's Canal, which corresponds to the vessel numbered 4 in Fig. 34.

The origin of the Anterior Venous return from the Ciliary Body is very extensive, being formed by the junction of innumerable branches to which the whole of the muscular plexus, as well as the vascular network of the Ciliary Process, contributes.

As these vessels perforate the sclera they anastomose freely with each other, as well as with the perforating branches of the Circulus Iridis. Many of the smaller Veins never completely perforate the Sclera at all, but act as connecting links between the larger vessels (Figs. 24 and 32).

The Anterior Uveal Veins.

The term Anterior Uveal Veins comprises the collective venous return from the Iris and Ciliary regions. The vessels from these areas, as they perforate the sclera at the pericorneal circumference, are closely associated with each other by anastomosing branches, including those sent to and received from Schlemm's Canal.

It is these linked Iris and Ciliary Veins which grouped together form an Anterior Uveal Venous system, as opposed to the Posterior System of the Venæ Vorticosæ, which latter system, as far as the anterior portion of the Uvea is concerned, constitutes merely an accessory channel, and not the sole route. The Posterior Uveal Venous system drains the Choroid, and while its radicles anastomose freely with branches from the Iris and Ciliary Veins, yet the most important physiological channels for the Anterior portion of the Uvea are the Anterior Uveal Veins, from which Schlemm's Canal extends as an offshoot.

The important fact, brought out by serial reconstruction, of the entire absence of Anterior Arteries makes it necessary to consider what and where are the vessels which take their place. On this point I have at present no direct anatomical evidence to bring forward, but, as will be discussed later, all physiological and pathological evidence points to the vascular supply to the Uvea being derived from the Posterior Ciliary Arteries, of which group of vessels the short ones are responsible for the arterial supply to the Choroid, while the long posterior Ciliary Arteries supply the Ciliary Body.

All the numerous vessels that perforate the corneal circumference are united together into one Venous system in close and direct association with Schlemm's Canal. This system of Anterior Uveal Veins, while draining the corresponding regions of the Uvea, is at the same time in free communication with the Posterior Uveal System of the Venæ Vorticosæ.

The significance of this anatomical disposition of the intra-ocular venous system is only apparent when the physiological fact, mentioned in discussing the eyeball as a closed sphere, is borne in mind—viz., that the intra-ocular pressure stands and varies with the intra-ocular venous pressure. For this relationship to hold good, it is essential that the aqueous have free access to veins (*vide infra*).

The channels available for aqueous resorption are the closely linked veins, comprising—

1. Schlemm's Canal, formed by tributaries from the Anterior Uveal Veins.
2. Iris Veins, access to which is through the Iris Crypts.
3. The Anterior Uveal Veins, as they cross the Supra-Choroidal Space.

There are no water-tight compartments in the eye, for the aqueous permeates freely through the iris and ciliary structures, thereby insuring that the same hydrostatic pressure exists in the supra-choroidal space as in the aqueous and vitreous chambers—in other words, throughout the whole of the intra-ocular contents. This pressure is the same as the intra-venous pressure, and it is only maintained at this level because of the free and ready contact between aqueous and veins.

The Superficial Pericorneal Capillary Plexus.

Every one of the Anterior Uveal Veins in its course through the sclera divides into anterior and posterior branches (V′ and V″ in Figs. 23 and 24, and also Figs. 28 to 32). This division takes place either on the surface of the Sclera, or in its parenchyma before the vessel has emerged on to the surface.

The posterior branches (V″, Figs. 23 and 24) join the muscular and other venous radicles of the episclera, while the anterior branches (V′) pass forwards and break up into a superficial pericorneal capillary plexus, which plexus is joined by the efferent vessels from Schlemm's Canal. The pericorneal capillary plexus thus belongs to the venous system. The necessity of such a venous plexus is obvious when it is realized that the cornea is nourished by the aqueous percolating through the open network of the Cribriform Ligament, and therefore a circum-corneal venous plexus is required to drain the corneal tissue, and not to nourish it (*vide infra*).

The Perivascular Lymph Sheaths.

The main vessels of the Anterior Uveal Venous System, including some of the largest of the afferent and efferent veins of Schlemm's Canal, are surrounded by perivascular lymph sheaths. The niche in which Schlemm's Canal lies is lined only on its outer side by a continuous endothelial lining, for on the inner the aqueous comes into direct contact with the sinus wall, while the endothelial cells are continuous with those lining the alveoli of the Cribriform Ligament.

While all the large Anterior Uveal Veins which directly traverse the sclera are invested with well-marked perivascular sheaths, their intercommunicating branches, as well as a number of vessels which do not themselves completely perforate the sclera, are not so supplied. The possession of a distinct perivascular sheath is, in fact, a characteristic feature of the larger vessels only.

The large perforating veins do not, under physiological conditions, fit their Scleral channels; and hence the necessity of having a reserve space filled with lymph ready to be displaced when the lumen of the vessels is increased, as, for instance, when intra-venous pressure is raised. In the case of the smaller vessels, whose walls are composed of only a single layer of endothelium, their lumen apparently fills up and occupies their scleral passages, but not so completely as to prevent aqueous diffusing along and between the vessel wall and the scleral canal. This fact is proved by the study of serial sections of cases of Irido-cyclitis, when pigment granules mark this out in a most clear and definite manner. The pigmentation round and outside the wall of these vessels conclusively demonstrates the presence of a lymph *space*, although there is no actual perivascular lymph sheath. A similar condition is normally seen in animals' eyes (dog, cat, etc.), in which one comes across patches of pigment in the perivascular lymph sheaths of the larger Anterior Uveal Veins, or between the vessel and the scleral wall in the case of the small vessels.

Comparative Anatomy of the Intra-Ocular Venous System.

In animals we find exactly the same analogous arrangement of the intra-ocular venous system as we have described in man.

The Veins of the Iris and Ciliary Body form a venous sinus, the *Circulus Venosus Hovii*. We have in the mammalia, too, the same arrangement of Anterior and Posterior Uveal Veins, with Schlemm's Canal formed as an offshoot from the former venous system.

The Anterior Uveal Veins, as in the human eye, send forward branches which form the superficial pericorneal capillary plexus, to which plexus efferent tributaries from Schlemm's Canal likewise contribute.

The analogous distribution of the intra-ocular venous system in man and in lower animals, associated with the conformity in the arrangement of the structures about the

Angle of the Anterior Chamber, are, I submit, convincing arguments of the correctness of the anatomical results furnished by reconstruction of serial sections. One finds throughout the Mammalia the same unity, such as modern science expects, in the general disposition of the intra-ocular structures, so that the anatomical conditions which I have particularly emphasized as existing in the human eye have in every case their counterpart in the lower animals.

One point in the comparative anatomy of the eye which I should like again to emphasize is the difference in the nature of the Iris tissue in animals and in man.

In animals the Iris tissue is relatively a dense fibrous structure in which crypts are absent, or poorly developed, while the human Iris is composed of a loose stroma with numerous crypts. This difference is an important one, as it has been concluded, as a result of experiments on animals, that the Iris is not a resorbing agent. This is certainly the case in some of the lower animals, but in man the Iris is more specialized, and its loose stroma, venous network, and open crypts, admirably fit it for its special function.

In animals the deficiency of the Iris as an accessory resorbing area is compensated by the facility with which aqueous can enter into the Supra-Choroidal Space owing to the absence of the obstructing scleral ring, as well as by the arrangement by which the Iris Root breaks up into pillars, so that the passage of Aqueous through the posterior part of the Cribriform Ligament into the Supra-Choroidal Space is quite unimpeded (Fig. 17).

SUMMARY OF UVEAL VENOUS SYSTEMS.

1. The Uveal Venous systems consist of—

    (*a*) Posterior Uveal Veins or Venæ Vorticosæ which drain the Choroid, and perforate the sclera behind the equator.

(*b*) The Anterior Uveal Veins from Iris and Ciliary Body, which perforate the sclera at the corneal periphery.

2. These two systems are closely linked together.

3. The Anterior Uveal Veins as they perforate the sclera freely anastomose among themselves, besides sending branches to, and receiving others from, Schlemm's Canal.

4. After perforating the sclera, the Anterior Uveal Veins form a superficial pericorneal capillary venous plexus.

5. Schlemm's Canal, being a secondary venous system derived from the Anterior Uveal Veins, must have an intravenous pressure just fractionally less than that within the Primary Veins of the Iris and Ciliary Body.

PART II

PHYSIOLOGY

The Nature of the Intra-Ocular Pressure.

According to the universal and prevailing opinion, the intra-ocular pressure is the product of a balance struck between inflow and outflow of the intra-ocular fluids. "The intra-ocular pressure is obviously a function of the contents of the globe. The solid part of the tissues being constant, variations in internal pressure must be due to changes in the amount of fluid."* As fluids are incompressible, the eyeball is necessarily regarded as an elastic sphere. The elasticity of the globe is, in fact, an indispensable corollary to the volumetric conception of the intra-ocular pressure, as only in consequence of its supposed elasticity could the eyeball accommodate any increased volume. The eyeball being regarded as an elastic globe, and variations in intra-ocular pressure being referred to variations in the volume of the intra-ocular contents, the maintenance of the form of the eye is naturally referred to the pressure exerted on the corneo-scleral envelope by its contents. Such, in brief, are the general and recognized views respecting the cause of the intra-ocular pressure and the nature of the corneo-scleral envelope. The assumption that "the intra-ocular pressure is obviously a function of the contents of the globe," although universally accepted, is one which is open to many and grave objections, of which the most obvious and telling is the fact that, if the intra-ocular and intra-cranial pressures are measured simultaneously, they are found to stand and vary together under all physiological and experimental conditions.

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* Parsons, "Pathology of the Eye," p. 1043.

Nature always works on the same general principles, however much the details may vary to suit particular physiological exigencies, and therefore it would be passing strange were the two pressures other than the same, considering the close embryological and anatomical associations between the contents of the cavities.

Thanks to the exhaustive researches of Leonard Hill,* the question of the nature of the intra-cranial pressure has been removed from the realm of speculation and theory to the definite region of experimental proof.

Leonard Hill has determined the important principle that in the closed and rigid case of the cranium the intra-cranial pressure represents the cerebral venous pressure, and varies directly with it, so that increase in the one always produces a corresponding and equal increase in the other. The intra-cranial and the cerebral venous pressures stand in much closer relation to the general venous than to the general arterial pressure, as they vary directly and absolutely with the pressure in the vena cava, but only in the direction of, and proportionately with, the pressure in the Aorta. This difference in the effect of the general circulation on the intra-cranial pressure is due to the fact that a rise in vena cava pressure makes itself felt at once by backward pressure on the cerebral venous capillaries, and therefore the intra-cranial pressure rises millimetre for millimetre with every increased millimetre of pressure in the vena cava; while, on the other hand, a rise of arterial pressure in the Aorta can only produce a proportional rise in the intra-cranial pressure, because between the Aorta and the cerebral veins lies the unknown and varying resistance of the arterioles. The intra-cranial pressure, as it stands and varies with the cerebral venous pressure, can be regarded as representing the pressure of the pumping force of the heart left over after the blood has been driven through and has overcome the resistance of the cerebral arterioles.

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* "The Physiology and Pathology of the Cerebral Circulation," 1896.

In a series of seven manometric observations made on dogs under Chloroform by Leonard Hill and myself, we found that not only did a brain-pressure gauge, and a similar suitable apparatus in the eye, give the same readings, about 10 millimetres Hg, but that the pressure registered in both rose and fell together the same amount under all circumstances.

In conducting such experimental observations, it is essential that great care be taken that the apparatus connected with the Anterior Chamber does not exert any pressure by its weight resting in any way on the eye, as discordant results will naturally follow.

In the last two experimental readings made, the procedure was much simplified by the use of the ingenious gauge devised by Leonard Hill for his pocket sphygmometer (Fig. 35). This gauge, although so simple in application, is perfectly accurate in its results, as can be proved by registering it against a mercurial manometer. In form the instrument resembles a clinical thermometer, consisting of a glass tube open at one end, and expanding into a small air space at the other end (B), which is formed of an inch of solid glass to hold the gauge by, so that the air space is not heated when held. Near the open end are a couple of side-holes (A), which mark the zero of the scale.

When the open end is dipped into water, the water rises by capillary attraction as far as the opposing-holes (A), where it forms a meniscus which registers the zero from which the gradations of the scale start. Having obtained a meniscus, a small length of stiff rubber tubing (D), connected with a hollow needle, is slipped over the open end of the gauge, so as to cover the side-holes, though before doing so the tubing and needle must be filled with normal saline solution. The gauge is now ready for use.

When the needle is inserted into the Anterior Chamber, the fluid in the needle, tubing, and the meniscus, form a single continuous column, which is pushed outwards against the column of air in the gauge, the resistance of which is registered in millimetres of mercury.

In the series of experiments performed, we always found that the same conditions that raised or lowered the intra-cranial pressure raised or lowered the intra-ocular pressure by exactly the same amount.

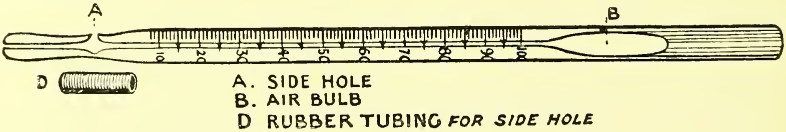

Fig. 35.—Leonard Hill's Sphygmometer Gauge.

Instead of a rubber tubing (D), the maker, Mr. J. J. Hicks, Hatton Gardens, has ground the end of the gauge for me so as to fit into a glass nozzle, like the end of a hypodermic syringe, on to which the needle is fitted. The gauge then forms one rigid instrument, which is much easier of application to the eye (see N, Figs. 43 and 44).

Neither the brain nor the eye possesses any mechanism by which it can be protected from changes in the general arterial or general venous pressure. Every variation in the general vascular pressure, and every muscular movement, is followed by corresponding changes in the cerebral and intra-ocular circulation, and therefore by simultaneous alterations in the pressure within the cranium and eyeball.

The response of the intra-ocular and intra-cranial pressures to the general vascular pressure is always prompt and simultaneous, except when the rise of pressure comes from a *sudden* rise in general venous pressure, in which case the ocular response is not quite so ready as the cerebral; but although not so prompt, it never fails to reach the same level. For example, when the abdomen is pressed upon, and the general venous pressure is thereby raised, the intra-cranial pressure mounts at once with the vena cava pressure; but the intra-ocular pressure, instead of rising *pari passu* with the intra-cranial, appears to lag just slightly behind it, so that a slight but appreciable difference of time elapses before it reaches the same height as the intra-cranial. That a sudden rise in general venous pressure should not produce such prompt response in the eye as it does in the brain is not surprising when one considers the extent and ramifications of the orbital veins into which the intra-ocular veins empty. In the eye, as compared with the brain, time is lost in transmitting a rise in general venous pressure, in consequence of the backward pressure having to be transmitted through the extensive orbital venous network before reaching and influencing the intra-ocular pressure. The response of the intra-ocular pressure is in consequence delayed in transmission, but not diminished in extent.

The experimental demonstration that the intra-ocular pressure stands at the same level and varies with the intra-cranial pressure, which latter in its turn is the same as the intra-cranial venous pressure, proves that *the intra-ocular pressure must be the same as intra-ocular venous pressure.* Further, however, as the pressures within the cranium and eyeball are the same in origin, nature, and level, therefore their enclosing coverings must be similar in nature, so that the fibrous corneo-scleral envelope represents an unyielding case with a fixed cubic capacity analogous in every respect to the bony cranium.

The intra-ocular pressure is, thus, not a question of volume of the intra-ocular contents, as is the opinion reflected throughout ophthalmological literature, by which preconceived notion all experimental data are biassed, but it is purely a question of pressure of a fixed volume in an unyielding case, the pressure being derived from the general circulation.

Variations in intra-ocular pressure can no more be the result of variations in intra-ocular volume than variations in intra-cranial pressure are the result of changes in intra-cranial volume. The conditions in the eye and brain are exactly the same, and what is true for the one applies with equal force to the other.

The equilibrium of pressure in the brain and eye insures the nervous and vascular elements connected with vision remaining throughout their whole course under the same pressure, and as the Lamina Cribrosa is not required to support any pressure, it is naturally the weakest spot in the corneo-scleral envelope.

The intra-ocular contents, like the intra-cranial, are contained in an unyielding case of limited cubic capacity, and the pressure of the fixed volume fluctuates because it represents the intra-ocular venous pressure, which in its turn depends on, and is influenced by, the general circulation. The intra-ocular pressure may therefore be defined in the same terms as the intra-cranial, as representing the pressure which remains in the eyeball after the force of the heart has been expended in driving the blood through the intra-ocular arterioles.

The reason why the intra-ocular pressure must be the same as the intra-venous pressure in the eye is obvious when we remember that fluids always remain at the lowest possible hydrostatic level. Now the intra-venous pressure is the lowest circulatory pressure in the eyeball, and therefore the intra-ocular fluids have no option, but their pressure must stand and vary with it, as being the lowest attainable hydrostatic level. The fact that the walls of the intra-ocular vascular system are elastic, and not rigid, make it impossible, from the purely mechanical standpoint, for the intra-ocular pressure to follow any other level but the intra-venous, so long as the eyeball remains a closed sphere.

Summary respecting the Physiology of the Intra-Ocular Pressure.

The conclusions to be drawn from the similarity in level found on simultaneous measurement of the intra-ocular and intra-cranial pressures may be summarized as follows:

1. The intra-ocular pressure is vascular in origin and nature, and stands and varies with intra-ocular venous pressure.
2. The intra-ocular pressure is influenced directly and absolutely by a rise in general venous pressure, but only relatively by increase in arterial pressure.
3. The intra-ocular volume is a fixed quantity.
4. The corneo-scleral envelope represents a rigid case.

The Corneo-Scleral Envelope.

The whole question of the nature of the intra-ocular tension hinges on the corneo-sclera being an unyielding case with a fixed volume, and therefore the conditions affecting the pressure of the intra-ocular contents under normal or pathological conditions cannot be clearly comprehended, unless this fact is kept prominently in mind.

That the corneo-sclera is an unyielding and rigid capsule with a fixed volume is a fundamental consideration which it will be necessary to examine and consider in some detail, particularly as the treatment of this question in the recent work by Parsons on the pathology of the eye must leave the reader in a condition of some bewilderment. It is, in fact, difficult to determine what final conclusions are meant to be drawn from such diametrically opposed assertions, as are conveniently made by the author to support theoretical arguments on the question of the intra-ocular circulation and pressure. On one page the statement is made that "the globe of the eye is practically rigid,"* which statement is flatly contradicted farther on by the assurance that, "unlike the cranium, however, the eyeball is not a rigid case;"† for "the effect of the elasticity of the walls of the eye has been already considered (*v.* p. 1040), and must be taken into consideration."‡ If, as directed, we refer back, we merely come across another series of conflicting statements, for now we find that "the elasticity of the walls of the eye is so great that the stretching under normal physiological pressure is minimal."§ The stretching is, in fact, "a quite appreciable amount,"|| even when the intra-ocular is raised from 19 millimetres Hg to 70 millimetres Hg.

With such a wealth of contradictions before us, it is impossible to extract any clear idea beyond a *reductio ad absurdum* as to the rigidity or otherwise of the corneo-scleral envelope. Apart, however, from any such deduction, one must take strong exception to the sense in which the term "elasticity" has been employed with reference to the eyeball—as exemplified, for instance, in the sentence quoted above, that "the elasticity of the walls of the eye is so great that the stretching under normal physiological pressure is minimal."

"Elasticity" does not signify the power of a structure to resist pressure, which is the meaning applied to that term by Parsons and other writers, but, on the contrary, it indicates that property whereby, after distortion, a body returns to its original form on the removal of a disturbing force.

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* Parsons, "Pathology of the Eye," p. 973.

† *Op. cit.*, p. 1046.

‡ *Op. cit.*, p. 1049.

§ *Op. cit.* p. 1041.

|| *Op. cit.*, p. 1042.

Elasticity of matter is defined by Lord Kelvin* as "that property in virtue of which a body requires force to change its bulk or shape, and requires a continued application of the force to maintain the change, and springs back when the force is removed, and, if left at rest without the force, does not remain at rest except in its previous bulk and shape." While according to Tait,† "Elasticity, in the correct use of the term, implies that property in a body in virtue of which it recovers, or tends to recover, from a deformation."

It is obviously impossible, with these definitions before us, to imagine that elasticity can be measured by means of resistance to pressure, for, as Gibson‡ reminds us, "a substance may have very small limits of resistance to stress, and yet be possessed of wide limits of elasticity. This is the true conception, equally removed from the two common misconceptions—firstly, that elasticity means resistance, and, secondly, that it is equivalent to distortability. Elasticity can only be estimated by the amount of recovery from distortion, and *the greater the distortion from which a body can recover, the greater is the elasticity of the body*." This is the very antithesis of the so-called "great elasticity" ascribed by Parsons to the corneo-scleral capsule.

If the walls of the eye were truly elastic in the proper sense of the word, then the eyeball, on being opened, should collapse like a punctured bicycle tyre. The eyeball does not yield before the normal intra-ocular tension because "the elasticity of the walls of the eye is so great," but because the rigidity of the connective-tissue covering is so marked. It is a question of an unyielding, not of an elastic, covering.

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* Kelvin, "Encyclopædia Britannica," 9th edit., vol. vii., p. 796.

† Tait, "Properties of Matter," p. 32.

‡ Gibson, "Misconceptions regarding Arterial Elasticity," *Lancet*, September, 1896.

The unyielding corneo-scleral envelope is analogous to a rigid case because its internal volume is a physiological fixed quantity. Rigidity in a *closed* receptacle containing fluids means that its cubic capacity can not be increased, and this applies as much to the fibrous coverings of the eye as to the osseous case of the brain. As the volume of the eyeball is a fixed quantity, variations in volume cannot take place to produce variations in pressure; but the pressure of the fixed volume varies within the closed and unyielding globe from the same cause, and under exactly similar conditions as in the cranium. It is the circulation that determines variations in intra-ocular pressure, which pressure, as we have shown, stands under all physiological conditions at the same level as the intra-cranial pressure. The important fact is thus established that the nervous elements connected with the act of vision, from Ora Serrata to Occipital Lobes, lie throughout their whole course under one and the same pressure. This pressure is not an absolute and fixed amount, but it represents the intravenous pressure within the closed cases of the eye and brain, and as such it varies within wide physiological limits depending on the general circulation.

The vascular conception of the intra-ocular pressure makes the absence, already detailed, of any hard-and-fast water-tight compartments between the various intra-ocular structures quite understandable, which would certainly not be the case were the intra-ocular pressure a question of volume, depending on the balance struck between secretion and resorption of aqueous.

Regulation of the Intra-Ocular Pressure.

The Aqueous in the Anterior Chamber comes into direct contact with the walls of the venous sinus of Schlemm's Canal, as well as with those of the Iris veins, while at the same time the fluid which passes into the Supra-Choroidal Space comes into association with the Anterior Uveal vessels as they cross that lymph space. The ready facility with which the Aqueous thus comes into direct contact with the intra-ocular veins accounts for the intra-ocular pressure being maintained at the level of the intra-ocular venous pressure. The elastic walls of the capillary veins cannot support any pressure, and hence, if the intra-venous pressure is raised, it is immediately transmitted to the intra-ocular fluid contents; and, *per contra*, if the pressure of the intra-ocular fluid contents is raised, the elastic venous walls are compressed till intra-venous pressure is raised to the same level. The elasticity of the venous walls precludes any dissociation of pressure between the intra- and extra-vascular fluids, so that their two pressures move together at the lowest attainable hydrostatic level of the intra-ocular circulation.

It has already been shown, by the reconstruction of serial sections, that Schlemm's Canal constitutes a secondary venous system formed by tributaries derived from the primary system of the Anterior Uveal Veins.

The fact, however, that Schlemm's Canal is a venous sinus does not in itself explain why this particular venous system should play such an important part in aqueous resorption, for anatomically Fuchs's Iris Crypts are to the Iris Veins what the Cribriform Ligament is to Schlemm's Canal: both crypts and perforated ligament allow of the free and ready access of aqueous to veins.

The physiological importance of Schlemm's Canal can only be appreciated when the nature and formation of this venous sinus are realized. Schlemm's Canal is a secondary venous system, and therefore its venous blood-pressure must be less than that in the primary and original venous areas of the Ciliary Body and Iris. The difference between the venous blood-pressure in Schlemm's Canal and that within the venous capillaries of the Iris cannot be very great; but however minimal, it is of great physiological importance to Schlemm's Canal, as it follows that the blood-pressure in this venous plexus is the lowest with which the aqueous in the Anterior Chamber comes into direct contact.

The intra-ocular and intra-cranial pressures are identical, and, as already discussed, represent the venous pressure within those closed spheres. In the eye, however, one can particularize still further, and state that *the intra-ocular pressure is equal to, and is always maintained at, the same level as the venous pressure within Schlemm's Canal.*

This deduction follows naturally in consequence of the venous pressure within the sinus being just less than that within the Iris Veins, and therefore the lowest circulatory pressure with which the aqueous comes into immediate contact.

Under physiological conditions the hydrostatic pressure of the aqueous can never be permanently maintained at any higher level than that within the sinus, because any increased aqueous pressure would act on the sinus wall of Schlemm's Canal, and, by diminishing its lumen, increase its venous pressure. As soon as the blood-pressure within the sinus becomes raised to the same level as that within the primary area of the Iris Veins, these latter are put on an equality with the former, and the aqueous will drain away as readily through the Iris Crypts and Veins as through the Cribriform Ligament and Schlemm's Canal. The accessory resorbing area of the Iris Veins is thus brought into action, and the increased hydrostatic pressure of the aqueous will diminish in proportion as it finds a route through the Iris Crypts into these veins. As the hydrostatic pressure of the aqueous then begins to go down, the pressure on the sinus wall of Schlemm's Canal is to the same extent relieved, and the intra-venous pressure within subsides once more below the level of that within the Iris capillaries, which are thus automatically excluded from participating further in aqueous resorption by their higher intra-venous pressure. Resorption of the aqueous cannot take place into a higher hydrostatic level, such as that presented normally by the Iris Veins, but only into one of the same level, which, under physiological conditions, is that existing within Schlemm's Canal. It is thus that Schlemm's Canal carries out the important part assigned to it in connection with aqueous resorption—a part which is quite inexplicable until the connections existing between the Iris Veins and Schlemm's Canal are appreciated.

The corneo-sclera being an unyielding capsule, and its total volume constant, one must regard the incompressible fluid volume in the aqueous chambers as complementary to the equally incompressible vascular volume; while, owing to the self-regulating and safety-valve action of the Iris Crypts and Veins, the pressure of the two fluid volumes is always kept identically the same.

Relation of the Intra-Ocular to the General Circulatory Pressure.

The close association and response of the intra-ocular pressure to changes in the general circulatory pressure have already been considered in a general way; but, in consequence of the important physiological and pathological conditions which hinge on this relationship, it is essential to discuss in some further detail the mechanism by which the pumping force of the heart induces and maintains the intra-ocular pressure.

Owing to the elastic nature of the circulatory apparatus, the arterial inflow of the eye is a constant stream, which at each cardiac systole is subjected to a wave of arterial pressure. This wave of pressure is transmitted directly to the intra-ocular contents, which in consequence manifest, on manometric observation, arterial pulsations, which pulsations may in turn be transmitted directly to the veins, producing the venous pulsation frequently seen on ophthalmoscopic examination in the veins near the disc.

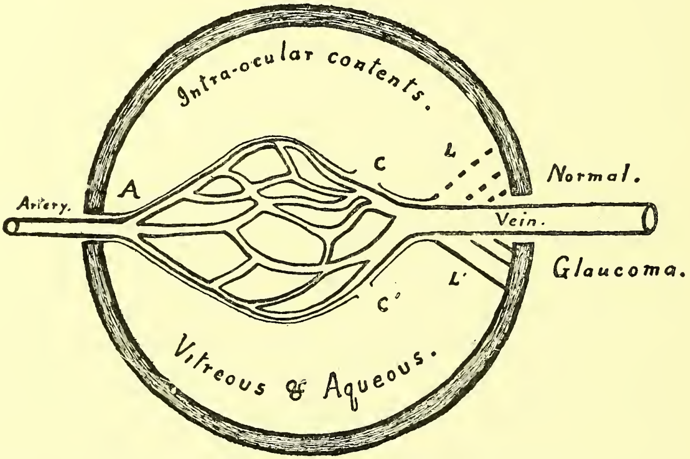

Fig. 36.—Schema illustrating the Circulatory Nature of the Intra-Ocular Pressure.

The upper half of the diagram represents the normal eye, with open meshes of Cribriform Ligament (L) and Iris Crypts (C). The globe of the corneosclera is an unyielding case, with cubic capacity strictly limited. The intraocular circulatory system is an elastic one. The fluid contents must be at the lowest hydrostatic level, which, in consequence of the free contact between the aqueous and veins at L and C, will represent the intra-venous pressure.

The pressure in the veins at the disc is naturally minimal, like that in Schlemm's Canal, and therefore the arterial pressure-wave transmitted to the intra-ocular contents (at A, Fig. 36) makes itself most evident in those vessels, or parts of vessels, in which the pressure is least (in the Vein, Fig. 36). The rhythmic waves of arterial pressure thus give rise to a negative venous pulse, in which the veins are narrowed, and the velocity of the venous outflow increased, when the arterial pressure-wave is at its maximum. The constant transference of this systolic wave of pressure through the intra-ocular contents helps in the nutrition of the intra-ocular contents by driving the Aqueous onwards, and preventing its stagnation in either of the Aqueous Chambers or in the intra-ocular tissues.

The arterial pulsatile pressure-wave can never produce such a compression of the veins as to lead to blocking of the circulation, because the pressure transmitted through the walls of the arteries (Fig. 36, A) to the intra-ocular contents must always be less than the pressure transmitted direct through the column of blood in the vessels. If, for instance, the intra-ocular pressure, as measured by a manometer, in the Anterior Chamber is 10 millimetres Hg, we know that this is the pressure which is transmitted direct through the continuous blood-column in the capillaries to the veins, for intra-venous and intra-ocular pressures stand together ; while, on the other hand, the pulsations registered by the same manometer are only a matter of a few millimetres Hg, which is an amount insufficient to overcome the venous pressure and occlude the veins.

If the arterial pressure rises, more pressure will certainly be transmitted through the arterial walls (at A) to the intra-ocular contents ; but simultaneously more pressure will be transmitted through the blood in the capillaries to the veins, so that it is impossible for the intra-ocular circulation to strangulate itself, as might otherwise be thought possible.

The intra-ocular contents are contained in a closed and unyielding case, and, in consequence of the free access of the aqueous to the veins, the intra-ocular and intra-venous pressures are the same. The intra-ocular fluids behave like all fluids retained in a closed sphere, in always tending to remain at the lowest possible hydrostatic level, which in the case of the closed eyeball represents the intra-venous pressure. On account of the elastic nature of the circulatory system of tubes, a rise of arterial pressure can only produce an increase in intra-ocular pressure in proportion as the resistance of the arterioles is overcome and the intra-venous pressure raised; while, on the other hand, a rise in general venous pressure by telling back on the intra-ocular veins will produce millimetre for millimetre a rise in intra-ocular pressure.

The whole question of the maintenance of the intra-ocular pressure and its relation to the general circulation resolves itself into the simple hydrostatic principle that fluids always tend to the lowest hydrostatic level, which in the closed sphere of the eyeball is that within the elastic venous walls. The application to the eye of this perfectly obvious hydrostatic principle completely alters the physical conception of the nature of the intra-ocular pressure, for if it holds good, then the current volumetric theory is absolutely untenable.

The Mechanism of the Intra-Ocular Pressure.

The mechanism by which the driving force of the circulation sustains the intra-ocular pressure can be admirably illustrated by a very simple working model, in which a flask filled with water represents the corneo-scleral envelope with its strictly limited capacity and incompressible contents, while a length of thin rubber tubing connected with a pair of the ordinary double spray bellows of commerce takes the place of the vascular system (Fig. 37).

All the essential features requisite to reproduce the mechanism of the intra-ocular pressure are thus represented—*i.e.*, a rigid case of fixed capacity through which circulates a fluid in an elastic system.

The flask I employed is one of 500 c.c. capacity, with a side-tube (L) connected by a small length of rubber tubing (T) with a Leonard Hill's manometer (M). The flask is closed by a rubber stopper (S), through which pass two glass tubes for inflow (A) and outflow (V). The inner ends of these tubes are connected by a yard of thin surgical

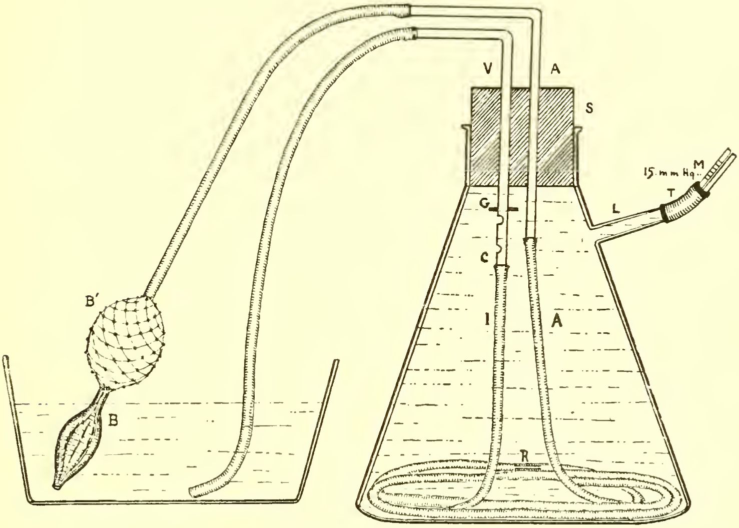

Fig. 37.—Working Model of the Circulatory Mechanism of the Intra-Ocular Pressure.

Flask with side-tube (L) to which is attached a Leonard Hill's manometer gauge (M) by a small length of thick rubber tubing (T). The flask is closed by a rubber stopper (S), through which pass two lengths of glass tubing (A and V), which are connected in the flask by a yard of thin elastic tubing.

To represent the resistance of the arterioles in this schema of the elastic circulation, its lumen should be reduced by a half by the insertion of a length of glass tubing (R) midway between A and I. The outflow tubing (V) has two perforations (C), which admit of free interchange between the circulating and contained fluids. The inflow tubing (A) is connected with a pair of double spray bellows (B and B′), which when worked drive the pressure in the flask up to 15 millimetres Hg, as measured by the manometer.

The conditions are exactly analogous to the normal eye—a rigid case, elastic circulatory system, and free contact between circulating and contained fluids. The contents pressure is not a question of volume, but of the lowest possible circulatory pressure, which, owing to the free communication at C, is naturally the same as the outflow or Venous pressure at V.

If the end of the rubber tubing is slipped up to the point G, so as to cover the openings C, the conditions in the flask are similar to those obtaining in the glaucomatous eye (see Fig. 39).

rubber tubing (of a bore of 4 millimetres), which lies coiled up at the bottom of the flask. The outflow tube has a couple of side-holes (C), each of a diameter of 4 millimetres, to allow of the free passage of the water in the flask with the fluid circulating through the rubber tubing. These holes take the place of the Cribriform Ligament, which associates the aqueous in the Anterior Chamber with the venous sinus of Schlemm's Canal.

The driving force of the circulatory pump is represented in the model by the double spray bellows (B), whose elastic reservoir (B′) converts the intermittent pumping into a continuous inflow stream. Though a continuous stream, its pressure naturally varies with each stroke of the syringe, and thus the conditions of the circulation through the flask are the same as those represented by the arterial circulation of the eye.

As the rubber tubing, representing the vascular system in the model, has a uniform internal diameter of 4 millimetres, there is nothing comparable in this arrangement to the resistance which the arterioles present to the circulation, and therefore to create such a resistance the lumen of the tubing should be reduced by the interposition at its mid-point of half an inch of glass tubing of 2 millimetres bore (R).

To work the model, the flask is filled with water, and the "vascular" tubing placed in coils at the bottom, care being taken to prevent kinking. The Leonard Hill's manometer (M), set at zero, is then connected with the side-tube (A) of the flask.

The air in the "vascular" tubing must next be driven out with a few strokes of the syringe, and when this is done the stopper can be pushed firmly home into the neck of the bottle. The volume displaced by the stopper finds a ready escape through the holes (C) of the outflow tube, and therefore the pressure in the bottle remains at zero.

As soon as the syringe is worked and water driven through the "vascular" tubing in the flask, then the conditions present are exactly similar to those which give rise to the normal intra-ocular pressure. We have a closed and rigid case with a fixed and incompressible fluid volume containing an elastic circulatory system, the fluid of which is brought into direct contact with the contents of the flask through the openings (C) of the outflow tube, which openings take the place of the Cribriform Ligament and Schlemm's Canal of the eye. With steady pumping the mean pressure in the flask registered by the manometer (M) is raised to 15 millimetres Hg, with an upward rise of about 5 millimetres Hg with each systole of the pump. The pressure in the flask is thus analogous in every respect to the intra-ocular pressure, which also manifests arterial pulsations on manometric observation. These oscillations represent the amount of systolic pressure transmitted through the walls of the rubber tubing to the fluid contents of the flask, while the pressure of 15 millimetres Hg represents what remains of the pumping force of the syringe after the greater part of the initial energy has been expended in driving the fluid through the elastic tubing.

The pressure within the flask is not a matter of volume, as the total volume is fixed at 500 c.c., and therefore cannot be increased; but it is purely a question of pressure derived from the force with which the syringe is worked.

Owing to the free communication at C between the water in the flask and that in the outflowing tubing (V), the pressure in the flask will always stand at the lowest hydrostatic level within the closed receptacle, which is that at which the circulatory fluid is being pumped out at the inflow.

The conditions which produce, maintain, and regulate the pressure in the flask are thus exactly the same as those which determine the pressure in the eye.

In the flask, as in the unyielding eyeball, the pressure is circulatory in origin, and derived from a pumping force, while the level at which it is maintained is the same as the outflow or return circulatory pressure, because that is the lowest hydrostatic level of the circulatory fluid. At whatever level the return circulatory pressure stands, the hydrostatic pressure within the closed receptacles of flask or eyeball must correspond to it, as being the minimum pressure.

As we have already seen, the intra-ocular pressure varies with a rise in general venous pressure, but only proportionately with an arterial one, as between the arteries and the intra-ocular veins lies the unknown resistance of the arterioles. Now, exactly the same condition exists in the flask in relation to its pressure.

If the outflow tube beyond the point V is pinched, the pressure in the flask rises millimetre for millimetre with the raised outflow pressure, while if we increase the inflow pressure by hard pumping the effect is only relative, depending on the extent to which the resistance of the rubber tubing is overcome and the outflow pressure increased.

The pressure in the eye and in the flask can thus both be expressed in the same terms as representing the hydrostatic pressure remaining after the pumping energy of heart or syringe has been expended in overcoming the elastic resistance of arterioles or rubber tubing. This pressure, as the lowest circulatory pressure attainable in the respective closed receptacles, is the one with which the pressure of the fluid contents will fluctuate.

Summary of the Mechanism of the Intra-Ocular Pressure.

1. The corneo-scleral envelope represents a rigid case with a fixed volume.
2. The walls of the vascular system are elastic.
3. There is free contact between Aqueous and Veins.

4. Fluids always remain at the lowest hydrostatic level; therefore the intra-ocular fluids are maintained and vary with the level of the intra-venous pressure, as being the lowest circulatory pressure in the eye.

5. Schlemm's Canal is a secondary venous system; therefore the pressure within it will be fractionally less than in the primary venous areas of the Iris and Ciliary Body.

6. The Intra-ocular pressure is the same as the intravenous pressure in Schlemm's Canal, as this is the lowest venous pressure in the eye.

Unity in Eye and Brain.

The association between the eye and brain does not only rest on the anatomical connection afforded by the embryological development of the eye as an outgrowth from the brain, but extends much farther in a close anatomical and physiological conformity of their respective contents in relation to their outer coverings.

The nerve and vascular elements subserving the visual function are throughout their whole course, whether in eyeball or cranium, subjected to a pressure which is the same in amount, in origin, and in nature. It is therefore not surprising that, to insure the even distribution of the varying vascular pressure, the same general arrangement of structures obtains within both unyielding envelopes. In both brain and eye the nerve elements are arranged as hollow spheres, which are protected from injurious contact with their firm cases by a water-bed as well as by a vascular bed (Fig. 38).

The brain mass is hollowed out by the lateral ventricles, while in the eye the hollow sphere of the retina encloses the vitreous chamber. The sub-dural and sub-arachnoid lymph spaces of the brain are represented in the eye by the supra-choroidal space. The fluid in these spaces, although minimal in amount, is of great physiological importance,

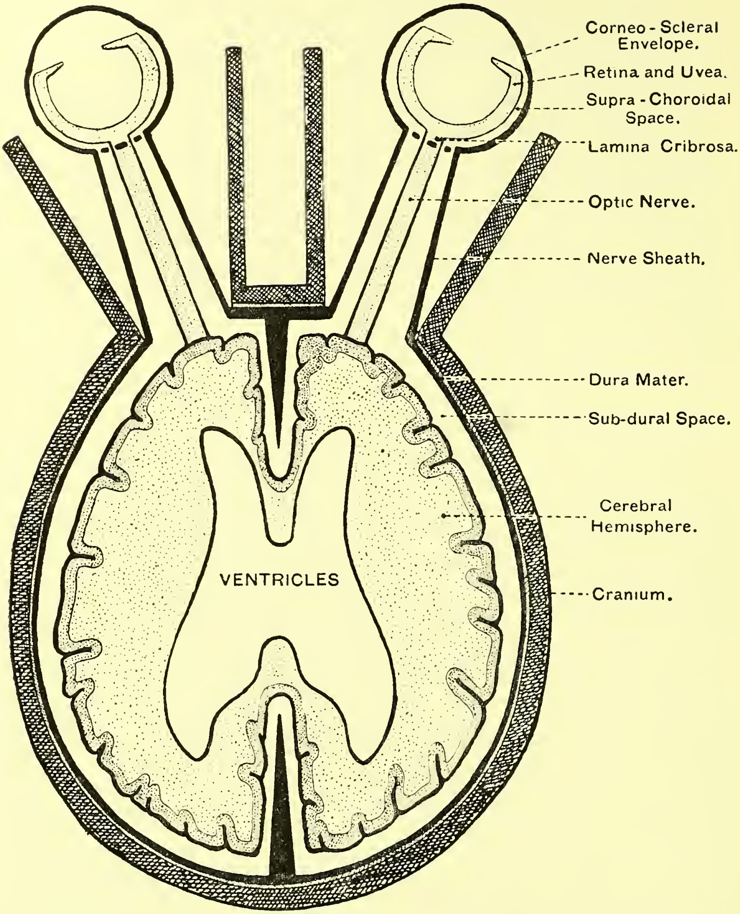

Fig. 38.—Analogous Disposition of the Intra-Ocular and Intra-Cranial Contents.

The nerve elements of the Eye (the retina) and of the Brain (the cerebral hemispheres) are arranged as hollow shells, containing respectively the Vitreous chamber and lateral Ventricles, the whole being enclosed by the unyielding outer coverings of the fibrous corneo-sclera and the bony cranium. The hydrostatic pressure within these two envelopes stands under all physiological conditions at the same intra-venous level; hence the structural weakness of the Lamina Cribrosa, as it is not required to withstand any pressure. To prevent injurious contact between nerve elements and their outer coverings are interposed the water-beds of the Supra-Choroidal and Sub-dural spaces, as well as the vascular beds of the Uvea and Pia Mater. The counterpart of the fluid reservoirs of the Aqueous Chambers of the Eye is represented in the Cranium by the basal Cisternæ Sub-arachnoidales.

as it ensures the even distribution of the hydrostatic pressure on all sides of the hollow spheres of nerve tissue.

These water-beds are in direct communication with the respective fluid reservoirs of the basal cisternæ sub-arachnoidales and aqueous chambers. These fluid reservoirs are necessary and very important formations. for otherwise, as the total volume within the cranium and eyeball is fixed, it would be impossible for changes in total vascular volume to take place. Variations in total vascular volume can only take place at the expense of the extra-vascular fluids, and hence the necessity of fluid reservoirs in the brain and eye whose volume can readily be displaced when the exigencies of the circulation demand.

The vascular bed is represented by the pia mater of the brain and the uveal tract of the eye. These vascular layers, together with the adjacent water-beds, effectively prevent all injurious contact between the nerve structures and their firm enveloping coverings.

The hollow globes of the cerebral hemispheres and retina lie literally embedded in fluid media, which exert an even pressure on the contents, equal to and varying with the intra-venous pressure. The Lamina Cribrosa is thus not required to withstand pressure. All the structures which pass through it lie throughout their whole course under identical pressures. This fact is of considerable clinical and pathological significance, for while under physiological conditions there is complete similarity in the amount of the hydrostatic pressure in the brain and eye, so that the pressures in both move together simultaneously, yet under pathological conditions there is at the level of the Lamina Cribrosa complete pressure discontinuity between them. Pathologically the pressure in either eye or brain can rise independently, and the effect of such dissociation is to produce characteristic clinical and pathological manifestations at the Disc (*vide infra*).

Comparison of the published analyses of the composition of the aqueous and of the cerebro-spinal fluid shows how exactly similar these two fluids are in their chemical properties and reactions. That this should be the case is not surprising, considering that both are formed under exactly analogous physiological conditions, and fulfil similar functions under similar hydrostatic pressures.

Both aqueous and cerebro-spinal fluid are clear colourless liquids, of a faintly alkaline reaction, and having a specific gravity of about 1008. Both fluids are described as containing a substance which is not sugar, but which, like sugar, reduces Fehling's Solution. Of dissolved solids they contain about 1 per cent., chiefly inorganic salts, of which the greater part is sodium chloride. Neither fluid coagulates spontaneously, as neither normally contains fibrinogen, which is only present after a paracentesis, when the fluid so obtained will clot on standing. The proteid percentage in both is very low, only about 1 part per 1,000, but it rapidly increases in both after tapping.

Gorup-Besanez[*] gives the proteid amount in cerebro-spinal fluid as 0·9 per 1,000, while Hoppe-Seyler's[†] analysis is a little higher, being 1·6 per 1,000. The published analysis of aqueous shows similar variations, as Gorup-Besanez[‡] estimates its proteid amount as 1·4, while Chavvas[§] and Lohmeyer[‖] agree in placing it at 1·2 per 1,000. When we consider the difficulty of collecting these fluids, owing to the small amount existing under normal conditions in the eye and brain, and that tapping increases their albuminous constituents, one must be struck by the fact of how closely they agree in their proteid percentage, averaging only about 1 part per 1,000.

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[*] See Schäfer's "Textbook of Physiology," vol. i., p. 24.

[†] Hoppe-Seyler, quoted in Landois and Stirling's "Physiology," p. 383.

[‡] See Schäfer, *loc cit.*

[§] See Schäfer, *loc. cit.*, p. 122.

[‖] See Schäfer, *loc. cit.*, p. 183.

In all essential details the aqueous and cerebro-spinal fluids show such a remarkable identity in their physiological relations and functions, process of formation, and chemical composition, that one is justified in adding another link to the chain of the points of resemblance, already noted as existing between the eye and brain, and stating that the aqueous and the cerebro-spinal fluids are the same—as much the same as the aqueous in one eye resembles that in the other eye.

Summary.

The parallelism existing between the intra-ocular and intra-cranial contents, illustrated in Fig. 38, may be tabulated as follows :

BRAIN.	EYE.
Outer Covering.	
Rigid bony cranium.	Unyielding fibrous corneo-scleral capsule.
Nervous Elements.	
Hollow Cerebral Hemispheres enclosing Lateral Ventricles.	Retina enclosing Vitreous Chamber.
Vascular Bed.	
Vascular Pia Mater.	Vascular Uvea.
Water-Bed.	
Sub-dural and Sub-arachnoid Spaces.	Supra-Choroidal Space.
Fluid Reservoirs.	
Basal Cisternæ Sub-arachnoidales.	Aqueous Chambers.
Physiological.	
Intra-cranial Pressure. Cerebral Venous Pressure.	Intra-ocular Pressure. Intra-ocular Venous Pressure.
Chemical Analysis.	
Cerebro-Spinal Fluid.	Aqueous Fluid.

The Function of the Iris.

In man the function of the Iris is not limited, as in most of the lower animals, to that of a mere mechanical shutter regulating the amount of light entering the eye, but it possesses an added specialized function in virtue of its numerous crypts, loose stroma, and rich vascular network.

As already discussed, the Iris venous system is a primary one, and therefore its vessels must have an intra-venous pressure fractionally higher than that within Schlemm's Canal, to which they act as accessory channels when circumstances demand such.

The Iris Crypts and Veins constitute auxiliary areas for aqueous resorption, and in consequence under conditions of increased intra-ocular pressure, whether pathologically or experimentally produced, the size of the pupil plays an important part. Dilatation of the pupil closes the mouths of the crypts, while contraction opens them, and so facilitates the access of the aqueous to the Iris Veins.

Under physiological conditions, with free access to Schlemm's Canal, and the Iris Crypts ever ready to assist as accessory absorbing channels, the conditions in the eye are ample to meet all contingencies which arise in consequence of the effect that variations in the general circulation have on the intra-ocular pressure.

If the arterial supply to the Iris and Ciliary Body is derived, as described by Leber, from a common trunk, the Circulus Arteriosus Iridis Major, and if, further, as is held by many writers, the Aqueous is a filtration and not a secretion, then there is no escaping from the obvious deduction that, as regards such a mechanical formation, the Iris vessels are in a much more favourable position than the Ciliary Capillaries. A filtrate from the Iris vessels would pass direct through the Crypts into the Anterior Chambers without having to be forced through the double layer of the Ciliary Epithelium.

The venous nature of the Circulus Iridis Major has already been demonstrated on anatomical grounds, but, apart from such direct evidence, it is physiologically impossible to endorse the arterial nature of this vascular circle without at the same time assigning an equally impracticable purpose to the ample arterial supply with which accepted anatomy would endow the Iris. Structure and function must harmonize, and hence the anatomical blood-supply to a part is always conditioned by the physiological activity of its tissue cells. Now, we have no evidence, commensurate with its supposed arterial supply, of manifestations of tissue activity in the loose Iris stroma; the only purpose of a large arterial supply would be connected with the secretion of aqueous, and to this all physiological and clinical evidence is opposed, for the aqueous formation is not affected in the slightest degree by the complete absence of the Iris, as, for instance, in cases of congenital or traumatic aniridia.

The anatomical proof already detailed of the venous nature of the vessels in question thus receives physiological confirmation. The vascularity of the Iris is, in fact, only intelligible when the venous nature of the Circulus Iridis Major is appreciated, and its connection with Schlemm's Canal recognized; for if the Iris is not a secreting structure, then a large arterial supply is uncalled for and unnecessary.

Secretion of Aqueous.

All clinical and experimental evidence points to the Ciliary Processes as the source of the Aqueous Fluid, though regarding the precise method of its formation there are, as in the case of the formation of lymph in the body generally, two opposing views. One school of thought considers the aqueous to be a filtration, while another regards it as a secretion formed by the active intervention of the lining cells of the Ciliary Processes.

The filtration hypothesis necessarily demands that there be definite compartments in the eye, in which the hydrostatic pressure on one side of the filtering membrane is greater than that on the other. That is to say, if the only force responsible for the Aqueous formation is a difference in hydrostatic level, then there must exist a pressure discontinuity between the tissue of the Ciliary Processes themselves and the intra-ocular fluids. It is certainly not sufficient to consider the difference in level between arterial pressure and the aqueous pressure, for the arterioles lie in a tissue enclosed by the filtering membrane, and therefore, if filtration is to be effective, the pressure of the tissues enclosed by the filtering membrane must be higher than the aqueous pressure. If such should not be the case, a filtration from the capillaries could certainly not pass through the ciliary epithelium, but would simply remain in the connective-tissue stroma, producing an œdematous condition of its structure.

Now, the aqueous finds a ready access into the Iris by way of Fuchs's Iris Crypts, and at the same time it passes into the Supra-Choroidal Space through the fasciculi composing the origin of the Ciliary Muscle. As the aqueous thus circulates on both sides of the filtering membrane, the essential mechanism for filtration, a difference in hydrostatic level between the two sides of a filtering membrane, is altogether absent. It is inconceivable that filtration can take place from the Ciliary vessels, through the Ciliary epithelial layers, and into the posterior aqueous chamber, when the hydrostatic pressure in the Supra-Choroidal Space, and therefore in the Ciliary Body, stands at the same level as that within the Aqueous Chambers.

The closer one looks into the conditions of aqueous formation, the less does one find them comply with those necessary for filtration, however simple such a mechanical process may appear at first sight. To comply with the mechanical requirements of filtration, the Iris surface would have to be devoid of crypts, and the existence of fluid in the Supra-Choroidal Space in some way discountenanced. The filtration theory omits to take account of the existence of the Iris crypts or of the Supra-Choroidal Space, and yet their presence cannot be ignored in connection with transmission of hydrostatic pressure.

Fluids are incompressible, and transmit pressure equally in all directions, while at the same time they always lie at the lowest possible pressure, which fundamental principles of hydrostatics must be, and are, followed by the fluid constituents enclosed within the unyielding corneo-sclera, as they are elsewhere in the physical world. The result is that the hydrostatic pressures of the vitreous, aqueous, and fluid in the Supra-Choroidal Space, all stand at the same level as the intra-venous pressure, because this is the lowest circulatory pressure in the closed sphere of the eyeball. So long as the eyeball remains a closed sphere, it is physically impossible for the aqueous to be a filtrate, because the same uniform hydrostatic pressure exists throughout the whole intra-ocular contents, so that a filtrate from the Ciliary capillaries would simply collect in the Supra-Choroidal Space without troubling to go through a double row of epithelial cells. When the eyeball is opened, however, the relationship of the intra-ocular to the intra-venous pressure is destroyed, for the intra-ocular pressure falls to zero, while the pressure of the circulation in the eye still responds to changes in the general circulation. Under these artifical conditions the fluid which is poured out is undoubtedly a true transudate, and consequently the amount obtained varies with the blood-pressure; but that this fluid represents the normal aqueous, either in mode of formation, composition, or amount produced, is an entirely unwarranted assumption. The fluid so obtained is a transudate, because the eyeball represents, not a closed, but an open sphere, and the alteration in its physical and chemical composition points to the altered conditions under which it was obtained. Normal aqueous only contains the merest traces of proteid, and does not coagulate spontaneously, while the fluid obtained after opening the eye may contain from 2 to 5 per cent. of proteid, and at the same time readily clots. Such experiments as those conducted by E. E. Henderson and Starling* certainly prove that the fluid which drains away from a cannula inserted in the Anterior Chamber is a filtrate, but beyond that their experimental findings have no further application, as the circulatory conditions in an open eye are perfectly different, and can in no way be likened to those existing in the normal and closed eyeball.

The anatomical and physiological evidence already brought forward in relation to the free communication between the aqueous on the one hand and the Iris stroma and the Supra-Choroidal Space on the other, and the equally important experimental proof of the vascular nature of the intra-ocular pressure and the parity existing between it and the intra-venous pressure, are all factors which make untenable the mechanical filtration hypothesis of the aqueous formation.

The absence of secretory nerves to the Ciliary Body of the eye, or to the Choroidal Fringes of the brain which secrete the Cerebro-spinal fluid, cannot be considered as any argument against the fluids in the eye and brain being formed by the active intervention of the lining cells of these structures, as only by secretory activity could fluid be collected in the reservoirs of the eye and brain, when the hydrostatic pressure within the

* Proceedings of the Royal Society, vol. lxxvii.

enclosed cavities is the same as the tissue and intra-venous pressure.

Treacher Collins* has described "Ciliary Glands," to which he ascribes the function of secretion; but these formations, apart from their unconvincing characters, do not even lie in the most vascular part of the Ciliary Body.

If these formations were true secreting "glands," both layers of the epithelial cells, and not merely the outer, should enter into their formation; and besides one would naturally expect to find them congregated at the Apices of the Ciliary Processes, as being the most vascular area, though it is just here that they are conspicuous by their absence.

That the Epithelium covering the Apices of the Ciliary Processes is most concerned with secretion is shown by the richness of the underlying fine capillary network, as well as by the progressive depigmentation which the outer layer of epithelial cells undergoes with advancing years.

The cells forming the inner stratum of the double row of the Epithelium of the Ciliary Body contain practically no pigment, while the cells of the outer layer, on the contrary, are pigmented throughout. Now this pigmentation of the outermost layer in a new-born child is very marked throughout its whole extent, but in a young adult, and progressively so the older the eye, the cells of the outer layer become less pigmented as they approach the Apices of the Processes, till in a senile eye the double row of epithelial cells in this region appears totally bleached (Fig. 56).

This progressive depigmentation of the outer layer of epithelial cells covering the Apices of the Ciliary Processes, coupled with the marked vascularity of this region, points to this area of the epithelial lining as being the one most intimately related to the secretion of Aqueous.

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* Treacher Collins, Trans. Oph. Soc. U.K., vol. xi., 1891.

SUMMARY.

1. The Aqueous is a secretion formed by the active intervention of the Epithelial cells lining the Apices of the Ciliary Processes.
2. Mechanical filtration is impossible, as there can be no difference in hydrostatic level on either side of the filtering membrane. The pressure of the fluid in the Supra-Choroidal Space must be the same as that in the Aqueous Chambers.

Resorption of Aqueous.

Reasons have already been given for stating that the hydrostatic pressure of the aqueous and the intra-venous pressure in Schlemm's Canal stand at the same level. The aqueous outflow therefore cannot be regarded as a filtration in the physical sense of the word, for, as in the case of the aqueous formation, the same hydrostatic pressure is to be found on both sides of what should represent the filtering membrane. The aqueous outflow is, in fact, not a filtration, but a diffusion or resorption, and the active and sole agents are veins, and only veins. The fundamental fact that aqueous resorption is determined solely by veins, and that Schlemm's Canal is a venous sinus, appears to be altogether lost sight of by the majority of experimental workers, and therefore the great mass of data and conclusions arrived at in connection with the attempts to measure the rate of aqueous resorption are proportionately valueless.

It is perfectly useless to estimate the amount of normal saline solution that can be driven through the Anterior Chamber at a constant pressure, and say that this must represent the amount of the normal aqueous outflow, because the essential premises on which such a deduction is based are erroneous; for, as already demonstrated, the intraocular pressure is not a question of volume, but of circulatory pressure.

In the eye and in the brain one can pour relatively any amount of fluid into their respective closed and unyielding coverings, but this no more represents the amount of aqueous formed in the one, than it does the amount of cerebro-spinal fluid in the other. The experiments only prove the extent of the accessory venous channels that come into action when required.

The experimental data obtained by driving fluid through *excised* eyes are still more worthless. Aqueous resorption is carried out at venous pressure by veins, and veins only, but when an eye is excised both the venous pressure and the intra-ocular pressure stand at zero. Now, when fluid is injected into an excised eyeball, the conditions present have no resemblance whatever to the relation of the aqueous "outflow" during life, because the intra-venous pressure in the enucleated eye is *nil*, and therefore the fluid passed in under pressure filters away. The process is admittedly a filtration, but under artificial conditions which are altogether unlike the normal physiological process when blood is circulating through the eye, and aqueous and intra-venous pressures stand at the same level.

In the closed and living eye the aqueous pressure stands and fluctuates with the hydrostatic level of the intra-ocular venous pressure, and therefore, in the absence of any difference in hydrostatic level between the two fluids, the aqueous outflow can never be a passive filtration, but must be an active resorption.

When the intra-ocular pressure rises, from whatever cause, then veins are compressed, and the venous pressure rises to the same level as the intra-ocular fluid pressure; and, similarly, when the intra-ocular venous pressure rises, the aqueous pressure mounts to the same height, for the elastic walls of the capillaries must transmit their pressure to the surrounding fluid.

Under all conditions, physiological or pathological, the aqueous pressure and intra-venous pressure are identical, so that aqueous outflow is a resorption into channels whose circulating fluid always lies under the same hydrostatic pressure.

The Aqueous in Relation to the Corneo-Scleral Envelope.

It is generally assumed that the lens is the only structure which the aqueous supplies with nutriment, for, according to modern teaching, the corneal tissue is not regarded as benefiting from the presence of the aqueous, as its nutritional supply is ascribed to an independent source derived from the Anterior Ciliary Arteries. As already shown, serial sections have failed to substantiate the presence of any such arterial vessels, and hence the necessity of modifying the current conceptions regarding the source of the nutrition for the cornea.

Experimental evidence also militates against the presence of a special arterial supply to the cornea, for while an incision concentric with the Limbus does not affect the corneal tissue deleteriously, yet section of the Long Posterior Ciliary Arteries or removal of the Ciliary Body is followed by degenerative changes* which can only be the result of changes in aqueous secretion, as the superficial, so-called "arterial" loops to the cornea are not interfered with, and therefore theoretically nutritional and degenerative changes should not follow.

The maintenance of the functions of the cornea and lens does not require the expenditure of energy, and therefore metabolism in both tissues is slow and sufficiently subserved by the aqueous, which, if sufficient for the one structure, is certainly quite sufficient for the other.

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* Wagenmann, *Archiv für Ophth.*, 1890 ; Siegrist, *ibid.*, 1900.

The layer of cells lining the corneal surface and the anterior lens capsule both act in the same manner by protecting their underlying tissues from the direct influence of the aqueous, the excessive passage of which has an injurious effect on their transparency. The changes which follow removal of the corneal endothelial layer or puncture of the lens capsule must be caused, not by any difference in the nature of the aqueous fluid which passes into the tissues, but must follow from the quantity which the tissues imbibe. In both cases we are dealing with highly specialized tissues whose condition of tissue existence is profoundly altered by the excessive passage of their nutrient fluid far beyond the limits of their physiological requirements. What applies to the transparent lenticular fibres applies with equal force to the equally transparent corneal fibres. It is not the diffusion of aqueous as aqueous that is harmful, but its excessive diffusion, which sets up tissue changes which lead to loss of transparency. Leber* has shown that the aqueous is prevented from passing directly into the Corneal Parenchyma by the layer of posterior corneal endothelial cells, but, as already discussed, the fibres of the Cribriform Ligament are continuous with the innermost lamellæ of the Cornea, and therefore the aqueous is able by this route to diffuse into the substantia propria of the Cornea. Now, the aqueous pressure is the same as the intra-ocular venous pressure, so that the passage of aqueous along and between the corneal lamellæ only takes place at the same level; that is to say, the process is one of diffusion or percolation of fluid at the same pressure, and not of filtration or passage of fluid from a higher to a lower hydrostatic level.

We need to distinguish very clearly between a process of diffusion and one of filtration of aqueous, because of late the possibility of obtaining permanent filtration through the cornea by means of specially-devised corneal incisions has been revived.

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* Leber, *Archiv für Ophth.*, 1873.

Such a means of permanently relieving the intra-ocular pressure is not, however, physiologically feasible; for, as one must again reiterate, the normal pressure in the eye is not a question of balance between the incoming and outgoing volume of intra-ocular fluids, but it is one of vascular pressure in a fixed volume, so that, when a wound leaks, the physiological relationship of parity between the aqueous and intra-venous pressure is lost.

As already discussed, the intra-ocular fluids have no choice, but must remain at the lowest possible hydrostatic level; so that, if permanent "filtering" cicatrices were possible, the result would be to produce a permanent dissociation between the venous and the intra-ocular pressure (*vide infra*, p. 203).

The process by which the aqueous passes outwards into the corneal stroma through the open network of the Cribriform Ligament is one of diffusion, and of diffusion only at the level of intra-venous pressure. The tissue tension of the corneo-sclera thus varies directly with that of the aqueous, which in its turn fluctuates with the intra-venous pressure within the eye.

This physiological association accounts for the bright flashing eye of high emotional states, and the dull cornea of low conditions of health. These changes in the corneal light reflex are induced by the corresponding variations in the general vascular pressure, which by affecting the intra-ocular tension produce similar and equal changes in the corneal tissue tension, and therefore in the light reflex which its surface gives off.

The corneo-sclera is analogous to a sphere of porous earthenware, but being lined at the back of the cornea by a continuous layer of endothelial cells, which act as a varnish or glaze, percolation cannot directly take place in this area.

Elsewhere, however, the sphere is not "glazed," so that the aqueous can diffuse through the Cribriform Ligament into the corneal tissue, and the fluid in the supra-choroidal space pass through the porous fibrous tissue of the sclera.

The superficial pericorneal network has already been shown to exist entirely of a venous plexus, the necessity of which is now apparent, as it is required to resorb the tissue fluids which have diffused through the corneal lamellæ, the superficial layers of which terminate by becoming incorporated in the loose stroma of the Limbus (Fig. 16).

The venous nature of the pericorneal network accounts for the injection accompanying Glaucoma and Iritis; for when the pressure in the intra-ocular veins is raised, it is obvious that the pressure in the venous ramifications forming the superficial plexus of vessels will likewise be raised, and engorgement and injection produced (see Figs. 23 and 24). If this plexus were formed by arterial branches, as figured by Leber, it would still require some explanation why the close vascular network should not normally produce a pink zone about the cornea. The so-called "arterioles" running to the plexus are ample to determine a blood-flow sufficient to fill the capillaries if they were really arterioles; but being venous branches, the blood-pressure within them is correspondingly low, and the capillaries remain proportionately empty (*vide infra*, p. 126).

The diffusion of aqueous at the Angle of the Anterior Chamber into the corneal stroma, and its passage into the supra-choroidal space, where it percolates through the scleral tissue, is an essential physiological process necessary for the maintenance of the nutrition of the tissue cells of the corneo-sclera.

As diffusion of the intra-ocular fluids takes place at the intra-venous level, the tissue tension of the fibrous framework of the eyeball varies directly with the intra-ocular pressure.

The fibrous tunic of the eyeball is not pushed out by the volume of the intra-ocular contents, for their volume is fixed, and the corneo-scleral capsule is an unyielding sphere whose tissue fluids lie under the same tension as the intra-ocular contents themselves; hence the eyeball retains its form solely in virtue of the rigidity of its framework, while its resistance to palpation depends upon and varies with the tension of its tissue fluids. The results are in absolute conformity and agreement with the circulatory nature of the intra-ocular pressure. What we recognize clinically as the normal intra-ocular pressure is not the resistance of an elastic tunic to the volume of its contents, but the intra-venous pressure of its fixed volume through the thickness and consistence of the fibrous coat of the eyeball.

To appreciate this fact we have but to realize that, in palpating over the open anterior fontanelle of the infantile cranium, the parts feel soft because we merely feel the intra-venous level of the intra-cranial contents through integuments which themselves offer little or no resistance. When the fontanelle is closed, the level and nature of the intra-cranial pressure are still exactly the same as before the bony cranial vault was completed, although now palpation reveals nothing but complete rigidity.

Now, whether in adult or infant the intra-ocular and intra-cranial pressures are the same, and therefore the difference which palpation reveals is merely due to the difference in rigidity of the outer tunics.

The maintenance of the form of the eye, as the maintenance of the shape of the cranium, is a function of the rigidity of the outer covering walls. The fibrous envelope of the eyeball is not as absolutely rigid as its analogue, the bony covering of the brain, yet it is more than sufficient to meet all physiological requirements in withstanding the venous level of the intra-ocular pressure, which, after all, is the same pressure as that which the cranial vault is called upon to support.

Relation of Aqueous Volume to the Vascular Volume.

The cubic capacity of the eyeball being fixed and the tissue volume constant, it follows that the total volume of intra-ocular fluids (vascular and extra-vascular) must be similarly constant. If the vascular volume increases, it can only be at the expense of the extra-vascular volume of the aqueous; in other words, the vascular and extra-vascular volumes are complementary to each other.

This is well illustrated by noting what takes place immediately after Paracentesis, when, as the aqueous escapes, the lens comes forward, the iris becomes hyperæmic, and in consequence the pupil contracts. These changes are the direct result of the corneo-scleral envelope acting as a rigid case with a definite and fixed cubic capacity, so that as the extra-vascular aqueous volume diminishes, its place is occupied by an exactly similar vascular volume.

The lens and vitreous are moved forward not because their volume expands under a relaxed pressure, as their volume is incompressible and fixed, but because the vascular bed of the uvea, on which they rest, becomes passively engorged to occupy the space of the escaped aqueous.

Paracentesis converts the rigid and closed case of the eyeball into a rigid and open one, and therefore the intra-ocular pressure falls to the same level as that within an open receptacle—*i.e.*, to zero. Since the intra-ocular and intra-venous pressures go hand in hand, when the eyeball is opened, and the intra-ocular pressure falls, the capillary venous pressure likewise falls—hence venous stasis and engorgement, in which the whole venous system of the uvea participates.

Occasionally when the diminution in volume is great, as after extraction or loss of vitreous, or when, owing to stiffening of the vessels, vascular compensation cannot take place sufficiently rapidly, indimpling of the Cornea or collapse of the Sclera may occur. Under these circumstances the creasing is the result of the diminution of the intra-ocular volume being greater than can be immediately compensated for by vascular dilatation, so that the atmospheric pressure causes the walls of the eye to cave in; and the spherical form of the eyeball is not re-established till the deficiency of the intra-ocular volume is made up, either by the entrance of an air-bubble into the Anterior Chamber or by the outpouring of a serous transudate.

So long as a corneal wound remains open there can be no intra-ocular pressure, as the fluid contents rest at zero till the margins of the incision are sufficiently agglutinated to withstand the normal intra-venous pressure of the intra-ocular contents. As soon as the wound can stand this amount of strain, the eyeball again becomes a physiologically closed sphere, which allows the Aqueous to collect in the Anterior Chamber at intra-venous pressure. The tension will, however, feel subnormal till consolidation of the wound is complete, as till then the sense of resistance imparted by the outer tissue is necessarily impaired by the "giving" of the lips of the incision (*vide infra*, p. 205).

SUMMARY OF THE PHYSIOLOGY OF THE AQUEOUS.

1. The Aqueous is a secretion formed by the active intervention of the epithelial cells covering the Ciliary Processes.

2. As the intra-ocular pressure stands at intra-venous level, filtration is not feasible, since the same hydrostatic pressure exists on both sides of the "filtering" membrane.

3. For the same reason the aqueous outflow is a diffusion or resorption at intra-venous level.

4. The nutrition of the Cornea is derived from the Aqueous diffusing through the origin of the Cribriform Ligament.

5. The corneal tissue fluid is resorbed by the superficial pericorneal venous plexus.

6. The tension of the corneo-scleral tissue fluids varies with the tension of the intra-ocular fluids.

7. The intra-ocular tension felt on palpating the eyeball is made up of the intra-venous level of the fixed intra-ocular contents plus the resistance of the corneo-scleral tunic.

The Intra-Ocular Circulation.

The fluid constituents of the intra-ocular circulation supply the necessary pabulum to the intra-ocular contents, while the force or energy with which the circulating fluids are driven through the vascular system of the eyeball is responsible for the maintenance of the intra-ocular pressure. For this composite function of the circulation the anatomical distribution of the vessels is admirably suited.

In the anatomical course of the intra-ocular vessels we find the explanation of a number of physiological, pathological, and clinical conditions which it is difficult to account for satisfactorily on any other grounds.

The intra-ocular vascular supply comprises the two systems of (1) the Retinal, (2) the Uveal Vessels.

1. The Retinal Circulation.—The Central Retinal Vessels run for some distance in the course of the Optic Nerve, and, as the intra-ocular and intra-cranial pressures stand and vary together under all conditions of the general circulation, it follows that changes in the Retinal circulation are a true index of the intra-ocular circulation and pressure, as the vessels, whether in the eye or brain, lie under the same intra-venous level of pressure throughout their course. This physiological parity of pressure in the eye and brain is a fact of considerable clinical significance (*vide infra*, p. 208).

The primitive crudeness of the volumetric conception of the intra-ocular pressure is apparent when one attempts to explain, according to this hypothesis, the relationship existing between intra-ocular and extra-ocular circulation. Parsons* states that "under normal conditions the fluid contents of the globe exert a pressure of 20 to 30 millimetres Hg above the atmospheric pressure, which may be regarded as the measure of the external pressure when the eye is at rest. The bloodvessels which enter the eye, therefore, are now subjected to an increased pressure of 20 to 30 millimetres Hg." This means nothing more or less than that the central artery of the Retina as it passes through the Lamina Cribrosa has suddenly imposed on it such a pressure as it enters the eye, while the vein with equal suddenness is released from this weight of pressure as it emerges from the eye. This sudden difference of 20 to 30 millimetres Hg pressure to which the venous wall is regarded as being subjected when in the eyeball, and from which it is released the instant it passes into the optic nerve, is an amount which is sufficient to meet the requirements of the most captious supporter of the filtration theory of lymph formation, and therefore the optic nerve for the first few millimetres behind the globe would surely be in a constant state of œdema.

No such violent and sudden transition of pressure does, however, take place, except under pathological conditions. In Glaucoma the intra-ocular pressure is really and truly raised above the intra-cranial, and therefore the pressure in the Retinal Vein is suddenly relieved as the vessel emerges through the Lamina Cribrosa, with the result that œdema and infiltration of the Optic nerve follows in consequence. The converse of this condition is met with in the pathological manifestation of choked disc which accompanies increased intra-cranial pressure (*vide infra*, p. 216).

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* Parsons, *op. cit.*, p. 1046.

2. **The Uveal Circulation.**—(*a*) *The arterial supply* to the Uvea is furnished, in the absence of any such vessels as Anterior Ciliary Arteries, by the Posterior Ciliary Arteries, and only by these vessels. These vessels may, in consequence, be more correctly described as the Uveal Arteries, of which the Posterior or short twigs supply the Choroid, while the Anterior or long branches transmit the arterial supply to the Ciliary Body and Processes, and therefore are associated with the Aqueous Secretion.

(*b*) *The venous return* from the Uvea corresponds closely to the physiological purpose of the arteries. For while the Anterior Uveal Arteries are associated with the Aqueous formation, the Anterior Uveal Veins are connected with Aqueous resorption. Similarly, the Posterior Uveal Arteries primarily supply the Choroid, and the corresponding Veins are chiefly concerned with its drainage.

The Anterior Uveal Veins return blood from the Iris and Ciliary Body by a series of vessels which, as they perforate the Sclera, freely intercommunicate with each other as well as with Schlemm's Canal, and then, after perforating the Sclera, these vessels send out loops to form a superficial capillary venous network in the Limbus to drain the corneal tissue fluids (Figs. 23 and 24).

The Posterior Uveal veins, or Venæ Vorticosæ, while draining the whole of the Choroid, yet receive numerous communicating branches from the Anterior Uveal Veins, which closely unite the two venous systems.

As the intra-ocular and intra-venous pressures stand at the same level, it is impossible for the uveal veins, as they emerge from the eye, to be normally subjected to any sudden release in their external pressure, amounting to between 20 and 30 millimetres Hg, such as Parsons in the above-quoted statement would have us believe.

As I have already shown, there exists at least one perforating vein to each millimetre of the corneal circumference, so that if these vessels were suddenly released from such a pressure the result would be permanent subconjunctival œdema. A further objection that I would raise against the unreasonableness of current views respecting the nature of the intra-ocular pressure is the fact that, if there existed Anterior Ciliary Arteries which formed, before entering the eye, the superficial pericorneal plexus, and which at the same time on entering the eye were suddenly subjected to an increased pressure of 20 to 30 millimetres Hg, it is perfectly obvious that the path of least resistance to the circulatory current would be on the surface, and the blood-flow would certainly choose this course rather than the deeper one. The result would be that the capillaries of the pericorneal plexus would always be filled, and the corneal margin would thus be outlined by a permanent pink zone. It is impossible to imagine how anything else could be expected under such circumstances, if they existed.

Take it how one will, the volumetric conception of the intra-ocular pressure makes a correlation between the extra-ocular and the intra-ocular circulation perfectly inconceivable; for the more strictly one reasons on the premises, the more absurd are the conclusions which one must arrive at.

Summary of the Intra-Ocular Circulation.

1. The intra-ocular and intra-cranial circulatory systems lie under the same intra-venous level of pressure.
2. The Uveal Vessels can be divided into (*a*) an Anterior, (*b*) a Posterior Uveal system.

3. The Anterior Uveal Arteries (Long Posterior Ciliaries) are the only arterial supply concerned with Aqueous secretion.
4. The Anterior Uveal Veins drain the Iris and Ciliary Body, and are concerned with Aqueous resorption.
5. The Posterior Uveal Arteries (Short Posterior Ciliaries) supply the Choroid.
6. The Posterior Uveal Veins drain the Choroid, though closely associated with the Anterior Uveal Veins.

PART III

PATHOLOGY

The Factors determining Primary Glaucoma.

In approaching the study of the Pathology of Glaucoma, the one physiological feature that must never be lost sight of is the circulatory nature of the intra-ocular pressure, as only when this conception is fully appreciated can the numerous and variable exciting causes responsible for a glaucomatous onset be co-ordinated with the equally numerous and variable clinical and pathological manifestations of the disease. The increased intra-ocular pressure in Glaucoma is no more the result of retention of intra-ocular volume than increased intra-cranial pressure is the result of increased intra-cranial volume. The physiological pressure in the eye, as in the brain, is vascular in origin and nature, and the pathological pressure is derived from the same source. Glaucoma is, in short, not a question of the retention of intra-ocular volume, but of intra-ocular pressure, the volume remaining constant.

The essential factors in Primary Glaucoma are two in number, and consist of a constant predisposing or mechanical agent associated with an exciting or vascular one.

The predisposing factor consists in sclerosis of the Cribriform Ligament, which raises a mechanical obstruction to the free access of aqueous to Schlemm's Canal, as well as to the Anterior Uveal Veins crossing the Supra-Choroidal Space.

The exciting factor is vascular, and depends directly on the relationship existing between the intra-ocular circulatory pressure and the general circulatory pressure.

The various clinical types and numerous pathological manifestations of Primary Glaucoma are the result of the combination in varying degrees of these two factors, both of which must co-exist to bring about and maintain a pathological rise of pressure.

Sclerosis of the Cribriform Ligament alone will not produce Primary Glaucoma so long as access to the Iris Veins is sufficient to maintain the intra-ocular pressure at intra-venous level, and the vascular factor remains in abeyance.

On the other hand, variations in general vascular pressure are by themselves ineffective without Sclerosis of the ligament; for the result of such will be merely temporary changes in pressure, easily righted in consequence of the extensive access which the aqueous normally has to the intra-ocular venous system.

The combination of these two factors will, however, effect a pathological rise of pressure, and the resulting clinical picture will vary as the one or other element predominates. In the acute congestive onset the vascular element is the paramount and dominant feature, while in the chronic non-congestive form of Glaucoma it plays an altogether subordinate and inconspicuous part. This same difference exists between the acute and chronic forms of all individual pathological conditions throughout the body, and Primary Glaucoma is no exception to the general rule, as in it, too, the only distinction between its extreme types is one of degree of action of the factors, and not a difference in their nature.

The wide diversities in the clinical and pathological manifestations of Primary Glaucoma can all be correlated as the direct outcome of the interplay of its two primary factors, the acute forms being characterized by "congestive œdema" of the tissues, and the chronic forms by their atrophy.

The Mechanism of Glaucoma.

Current hypotheses of Glaucoma ascribe the increased pressure to a retention of intra-ocular fluids, thereby implying an increased volume such as is impossible within the unyielding walls of the eyeball.

Glaucoma is not produced by a retention of intra-ocular volume, but by the retention of intra-ocular pressure, which pressure, as in the case of the normal intra-ocular pressure, is derived from the circulation. How such a retention of pressure is brought about can be most satisfactorily and easily demonstrated in the working model already constructed to illustrate the mechanism of the normal intra-ocular pressure (Fig. 37).

In this model, when the syringe is worked, the pressure in the flask is derived from the force with which fluid is driven through the elastic tubing, and this pressure, owing to the free communication (at C) between the water in the flask and that circulating in the "vascular" tubing, will always stand at the same level as that in the outflow tubing (V), as being the lowest hydrostatic pressure in the flask.

The conditions in the flask, as already discussed, are comparable in every way to those in the eye, in which the intra-ocular pressure similarly stands at the lowest possible circulatory level, which is the intra-ocular venous pressure.

To make the model correspond to a glaucomatous eye (Fig. 39), the elastic tubing at the outflow end is slipped over the openings (C) to the point G, so that the connection between the fluid in the flask and the water circulating in the tube is cut off. The condition present in the flask will now be similar to those in Glaucoma, in which the intra-ocular fluids are shut off from Schlemm's Canal by sclerosis of the Cribriform Ligament. The result in both cases is the same, as the fluid contents are prevented from diffusing into the circulating fluid.

The elastic circulatory systems of both flask and glaucomatous eye are contained in unyielding cases, and at the same time surrounded by fluid, and therefore incompressible, contents which act as a solid mass, the effect of which is to convert the elastic walls of the circulatory systems into rigid ones.

In a rigid system of tubes the outflow pressure approximates to the inflow pressure, while in an elastic one the outflow pressure represents only the pressure left over after the resistance of the elastic walls has been overcome. The more an elastic system of tubes approximates to a rigid system, less energy is expended in overcoming resistance, and therefore the higher is the outflow pressure.

When the fluid contents in the flask or eye are prevented from direct access to the outflow or return circulation, their incompressible volume tends to convert the elastic walls of the circulatory system into rigid ones, with the result that the outflow pressure mounts up towards the level of the pressure of the inflow or arterial side. The fluid contents in the flask or eye still lie at the lowest attainable hydrostatic level, which is that of the return circulation; but now, as the vascular elastic system of tubes is analogous to a rigid system, this outflow circulatory pressure is raised, and with it *pari passu* the contents pressure in the flask or eye is increased. The result is a vicious circle in which the pressure of the fluid contents causes the elastic walls of the circulatory systems to assume more and more the properties of a rigid system, and in consequence the outflow (venous) pressure rises higher and higher. This increase in its turn reacts on the pressure of the fluid contents, whose pressure must always keep pace with the outflow pressure.

The conditions which occasion the maintenance of increased pressure in flask or eye are induced by causes in which the circulatory pressure alone is the mainspring, and in which considerations of volume, beyond that of a fixed total volume, do not enter.

The contents pressure rises because the elastic walls of the circulation are converted into rigid ones, and therefore the outflow circulatory pressure approximates the inflow or arterial pressure. The lowest attainable hydrostatic level is that of a rigid system of tubes.

To work the model to represent Glaucoma (Fig. 39), the rubber tubing is slipped over the side-holes (C) of the outflow tube, and the flask filled with water. The stopper (S) is then driven home, and the volume of water so displaced escapes through the side-opening (L and T). Leonard Hill's manometer gauge set at zero is next slipped into position into the tubing (T). Now, as soon as the syringe (B) is worked, the pressure in the flask begins to mount up to about 60 millimetres Hg—that is to say, to a pressure four times that obtained when the fluid contents were in free communication with the outflow circulating fluid. This high pressure is not due to any retention of volume in the flask, but is simply the result of the conversion of the elastic tubing into a rigid system, and of the difference in the mode of transmission of the circulatory force.

The mechanical principles involved here are exactly the same as in the Eye with Glaucoma, hence the importance of appreciating how the changes in the transmission of the circulatory pressure are induced. In the elastic tubing of the " Glaucoma " flask the pumping force is transmitted at A to the incompressible fluid contents, which contents, having no direct contact with the circulatory fluid, must exert pressure on and through the elastic wall of the distal or outflow part of the system (I). Here the circulatory pressure is naturally lowest, and therefore the elastic walls are compressed and made similar to rigid walls. The outflow-pressure is thus raised to correspond

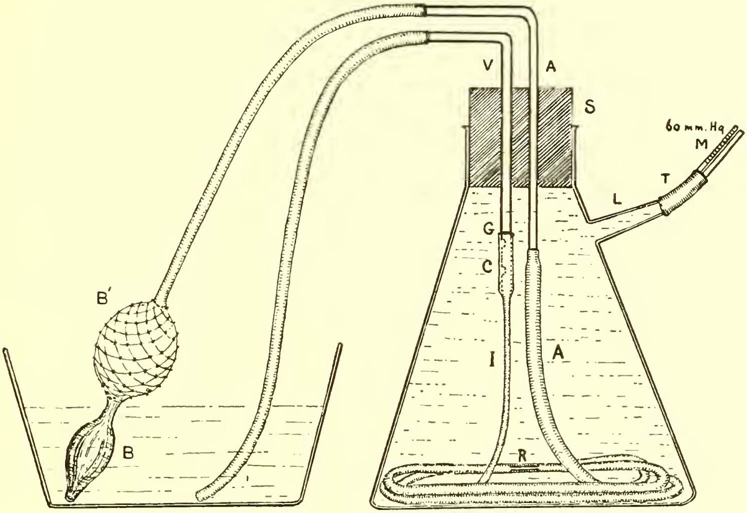

Fig. 39.—Working Model of the Mechanism of the Pressure in Glaucoma.

The model is the same as that illustrated in Fig. 37, only the end I of the rubber tubing covers the perforations (C) of the outflow tube (V); the circulating fluid is thus completely shut off from the fluid in the flask, which thus acts as a rigid mass, converting the elastic walls of the tubing into rigid ones. The nature of the circulatory flow is thus altered, and in consequence the relative amount of fluid in the different areas of the tubing changes, although the circulating volume within it remains the same, as the cubic capacity of the flask is fixed and the contained volume cannot vary, as it is completely shut off from the circulating fluid. The circulatory flow through the flask is of the nature of a rigid system, and therefore the return (venous) circulatory pressure at V rises towards the inflow (arterial) pressure at A, and the tension in the flask mounts to 60 millimetres Hg. This represents the lowest circulatory pressure in the flask.

with the contents-pressure, while at the same time the whole circulatory flow approaches that of a rigid system.

Now the cubic capacity of the flask is fixed, and the fluid contents volume constant, and therefore compression of the outflow (venous) part of the tubing takes place only at the expense of dilatation of the inflow (arterial) tubing; that is to say, the relative amount of fluid in the various parts of the circulatory system is altered.

So long as regular pumping is maintained, the pressure in the flask registers about 60 millimetres Hg, while if continued with diminished force the pressure registered is proportionately less. If the pumping force is increased, the pressure in the flask may be raised to about 80 millimetres Hg, at which pressure the circulation through the tubing will be brought practically to a standstill, as the pressure inside the flask will equal the pumping force with which the fluid is driven through the tubing.

In flask or eye, maintenance of the difference in the level of pressure existing under normal conditions and in Glaucoma depends on the causes whereby the circulation is in the former condition carried through an elastic system, while in the latter it approximates that in a rigid system; in which less of the driving energy is expended in overcoming the resistance of the vascular walls, and therefore the outflow (venous) circulatory pressure approaches the inflow or pumping force.

Whatever the outflow or venous pressure may be, the pressure of the fluid contents must be maintained at the same level—namely, the lowest hydrostatic pressure existing within the enclosed space. In Glaucoma the intra-ocular pressure is still the same as the venous pressure, but the venous pressure is raised because the circulation is reduced to a rigid system; and in this way the intra-venous pressure approximates the arterial circulatory pressure. The lowest circulatory pressure in Glaucoma is that of a rigid, and not of an elastic, system of tubes; hence the sustained high pressure in the eye.

SUMMARY OF THE MECHANISM OF GLAUCOMA.

1. The free contact between Aqueous and Veins causes the intra-ocular pressure to be maintained at the normal intra-venous level, as this is the lowest circulatory pressure.
2. In Glaucoma the contact is diminished, and the intra-ocular fluids, being contained in an unyielding capsule, act as a rigid volume which, operating through the tissues, compels the intra-ocular circulation to run in rigid lines.
3. In a rigid system the outflow pressure is always higher than in a similar system of elastic tubes.
4. In Glaucoma the lowest circulatory pressure is that in a rigid system, and therefore the intra-ocular pressure is maintained at a correspondingly high level.

The Predisposing and Constant Factor in Glaucoma.

The sclerosis of the Cribriform Ligament in Primary Glaucoma is simply a continuation, culminating in a pathological excess, of the process of fibrosis which the Ligament undergoes throughout life. Physiologically this sclerosis is the direct result of the Cribriform Ligament acting as the ligament of origin of the Ciliary Muscle, and therefore this process is at first strictly limited to the innermost or Ciliary Fibres. The association, so frequently asserted, between Glaucoma and Hypermetropia is undoubtedly due to the influence which the constant and excessive action of the Ciliary Muscle in this refractive condition of the eye must have on the Cribriform Ligament in tending to produce an early and marked sclerosis.

The outmost strands of the Cribriform Ligament next to Schlemm's Canal, being incorporated in the Sclera, are not affected by muscular action, and therefore do not undergo any very marked changes till the presbyopic years have been reached. In this division of the Ligament, there can be little doubt that the process of sclerosis is initiated by the same agencies, which, associated with advancing years, induce sclerosis elsewhere in the tissues of the body. These agencies include such well-recognized conditions as gout, rheumatism, auto-intoxication, syphilis, and many other constitutional diseases unnecessary to enumerate, all of which have the one common tendency of exciting fibrotic changes.

In a senile eye the individual strands of the Ciliary portion of the Cribriform Ligament attain a thickness, exclusive of cellular elements, of between 5 and 6 micros. In Glaucoma the size of these strands is greater, as they generally appear to reach the uniform thickness of 7 micros. The meshes of the network are thus proportionately reduced.

Sclerosis of the Cribriform Ligament is in itself the *predisposing* factor in Glaucoma, the effect of which is to obstruct the free passage of Aqueous to Schlemm's Canal. The obstruction raised by Sclerosis, it is essential to note, is not necessarily absolute, but relative, being relative to the exciting or vascular factor, thus accounting for the fact that, while in some cases the Ligament appears as a dense, compact fibrous structure, in others the total changes present may not appear to be much more marked than in a normal eye of about the same period of life.

The progressive sclerosis of the Cribriform Ligament reaches pathological limits as soon as the meshes of its open lattice-work become so reduced by the thickening of the individual strands that the ready passage of Aqueous to Schlemm's Canal is impeded.

The conditions which determine and maintain a pathological rise in intra-ocular pressure, as well as the resulting clinical manifestations, depend directly on the extent of action of the vascular factor; in other words, the different clinical types of Primary Glaucoma differ *inter se* in degree, but not in kind. It is the play and interplay of the constant sclerotic factor with the variable vascular factor which give to each case of Primary Glaucoma an individuality that precludes the too close restriction of the disease into distant forms and types.

The Aqueous normally comes into contact with (1) the Venous Sinus of Schlemm's Canal, (2) the Anterior Uveal Veins traversing the Supra-Choroidal Space, and (3) the Iris Veins; but as Sclerosis of the Cribriform Ligament increases, the first two routes are obstructed, leaving only the last. It is this diminution in the range of access of Aqueous to the intra-ocular veins which alters the conditions affecting the intra-ocular circulation, and converts what would be otherwise merely physiological and temporary into pathological and permanent increase in pressure.

The effect of Sclerosis of the Cribriform Ligament on the intra-ocular circulation is exactly the same as that obtained in the "Glaucoma" Flask (Fig. 39) by slipping the rubber tubing over the openings C, and so shutting off the fluid contents from the circulating fluid.

The Exciting and Variable Factor in Glaucoma.

The vascular changes determining acute Glaucoma are the direct outcome of alterations in the general circulatory pressure; for, as already stated, neither the eye nor brain possess any protective mechanism by which they can be secured from such changes. A rise in the Vena Cava pressure produces millimetre for millimetre a corresponding and equal rise in intra-ocular and intra-cranial pressures, while increase of pressure in the Aorta causes a rise of pressure in the eye and brain, whose exact amount depends on the varying resistance of the arterioles.

In the closed corneo-scleral capsule the normal intra-ocular pressure is an ever-varying one, and in consequence it is essential, for its maintenance at intra-venous level, that the venous areas available for the aqueous be sufficiently wide and extensive to meet all physiological contingencies.

Under normal and physiological conditions, no continuous and pathological increase of pressure can be transmitted by the intra-ocular fluids, as the aqueous cannot be retained in the Anterior Chamber at a pressure above the intra-venous pressure. The pressure in both veins and aqueous must go hand in hand, because the intra-venous level is the lowest hydrostatic pressure within the eyeball, and the contact between the two is so free.

With Sclerosis of the Cribriform Ligament, however, the Iris Crypts, instead of being accessory passages, are now the only ones through which the Aqueous can come into direct contact with the intra-ocular venous system, and if at the same time the minimum circulatory pressure in the eye is raised by increase in the general vascular pressure, the intra-ocular pressure is raised also with serious results. The contact between aqueous and veins is insufficient, and the raised pressure of the intra-ocular fluid contents, instead of acting directly on the venous walls, does so only indirectly through the uveal tissues, so that the intra-ocular circulation is forced into rigid lines (Fig. 40).

An acute glaucomatous onset is thus the direct consequence of the physiological association between variations in the intra-ocular pressure and the general arterial and venous pressures, which sclerosis of the Cribriform Ligament renders pathological by hindering free and ready contact.

Given the primary restriction and curtailment of the range of direct contact between aqueous and the intra-ocular venous system, all such conditions which act by raising the general vascular pressure either on the arterial or venous side will precipitate Glaucoma. Viewed in this light, Primary Glaucoma is a disturbance of the circulation in the eye, induced by corresponding changes in the general circulation. It is this way that such varying exciting agents as psychic disturbances, congested portal system, or overloaded *primæ viæ*, etc., exert their influence by increasing the general circulatory pressure, and so, simultaneously, the intra-ocular pressure.

Increase in General Arterial Pressure produces a rise in

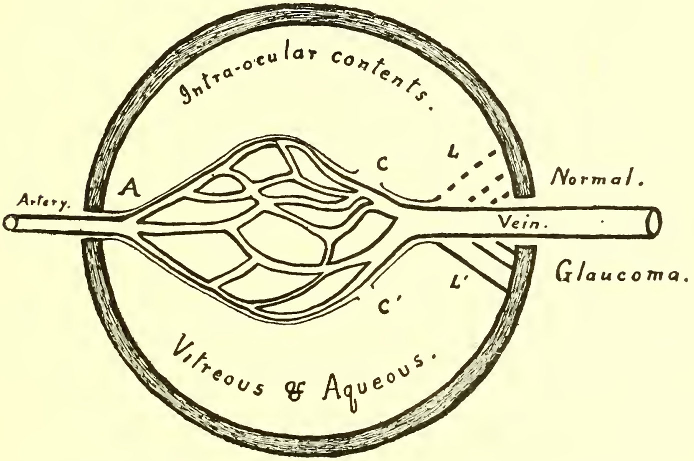

Fig. 40.—Schema illustrating the Circulatory Nature of the Intra-Ocular Pressure.

The eyeball represents an unyielding case with a strictly limited cubic capacity.

In the Normal Eye (upper half of diagram) the meshes of the Cribriform Ligament (L) are open. The intra-ocular contents lie at intra-venous pressure, as such is the lowest attainable hydrostatic level. The circulation is conducted through an elastic system.

In the Glaucomatous Eye (lower half of diagram) the Cribriform Ligament (L′) is sclerosed, and its meshes narrowed or occluded altogether, leaving Iris Crypts (C′) as the only direct route for contact between Aqueous and Veins.

The incompressible intra-ocular contents of the Vitreous and Aqueous then tend to act as a rigid mass, which converts the elastic walls of the circulation into rigid ones. The more complete this conversion, the more the venous pressure approximates the higher level of the arterial pressure, and with this change the intra-ocular pressure follows suit. The contents pressure will still represent whatever level the venous pressure may be, as such must always remain the lowest circulatory pressure, but the venous outflow is at a higher hydrostatic level—hence the maintenance of the glaucomatous pressure.

the intra-ocular pressure, the amount of which depends on the extent of the resistance of the intra-ocular arterioles, and the increased circulatory pressure which they allow to be transmitted direct to the veins. But so long as contact between the Aqueous and Veins is extensive enough, no pathological results will follow, as the circulation will still be conducted through elastic walls; and while the intra-ocular pressure is raised in unison with the venous pressure, the effect is merely a physiological change in the amount of the intra-ocular pressure. In an eye, however, with sclerosis of the Cribriform Ligament, the effect of such a rise will produce serious pathological consequences; for as the intra-venous pressure rises, the intra-ocular pressure naturally follows to the same height, but, in consequence of the diminished range of contact between the intra-ocular fluids and the intra-ocular veins, the result of the increased intra-ocular pressure is to convert the circulation in the eye into a rigid system.

Increase in General Venous Pressure is one of the most potent exciting causes of acute Glaucoma. The cause of this lies in the intimate relationship between the venous pressure in the abdominal area and the venous pressure of the cerebral and ocular circulations, whereby a rise in the former area tells directly on the latter venous area, producing a similar rise in intra-ocular pressure. If the passage of aqueous to the intra-ocular veins is free enough, such a rise merely produces a physiological change in the intra-ocular pressure, and the intra-ocular circulation will still be conducted through elastic walls; but it is otherwise in an eye with a tendency to Glaucoma—*i.e.*, with sclerosed Cribriform Ligament, and therefore diminished access of Aqueous to Veins. In such an eye, the effect of a rise of venous, and therefore intra-ocular, pressure is to convert the circulation into a rigid system, with the result that, as the outflow or venous pressure of such a rigid system rises, the general contents pressure rises too, and is sustained at a pathological level because of the altered nature of the circulation.

In Glaucoma, whether the initial impulse comes from the arterial or the venous side of the general circulation, the ultimate effect on the intra-ocular circulation and pressure is exactly the same. The circulation is forced to run in rigid lines, and therefore the intra-venous pressure is maintained at a higher level, and with it the intra-ocular pressure.

As the elastic intra-ocular circulation approximates to a rigid system, the effect, as in the brain under analogous conditions, is to increase the velocity of the blood-flow, and to change the relative distribution of the blood in arteries, capillaries, and veins; for little arterial energy is spent now in overcoming the resistance of the intra-ocular arterioles, but much in overcoming the resistance of the venous exit. In Chronic Glaucoma the effect of this is very striking in the extensive venous formation in the Iris, and in the number and size of the Anterior Uveal Veins.

SUMMARY.

The view that Primary Glaucoma is the product of retention of intra ocular volume has obtained for so long, and has received such truly universal recognition, that I am constrained, even at the risk of tedious repetition, to emphasize the principles involved by the circulatory nature of the Intra-ocular pressure under physiological or pathological conditions, by restating them as follows:

1. *General Principles.*

1. The eyeball is an unyielding capsule of fixed cubic capacity.

2. The circulation is conducted in an elastic system of vessels.

3. The intra-ocular pressure is derived from the circulatory pressure.

4. The intra-ocular pressure stands and varies with the intra-venous pressure in the eye, as the lowest circulatory pressure within that closed sphere.

5. The intra-venous, and therefore the intra-ocular, pressure is influenced by all changes in the general arterial and general venous pressure.

6. The conditions of the circulation and pressure in the eye and brain are the same.

2. *Comparison of the Conditions in the Normal Eye and Glaucoma.*

NORMAL EYE.	GLAUCOMA.
1. Aqueous is in free communication with (*a*) Schlemm's Canal. (*b*) Supra-Choroidal Sphere. (*c*) Iris Veins.	1. Sclerosis of the Cribriform Ligament, and therefore Aqueous only in contact with Iris Veins.
2. The intra-ocular circulation is an elastic system.	2. The intra-ocular fluids act as a rigid volume, which, by transmitting their pressure through the tissues, compel the circulation to run in rigid lines.
3. The intra-ocular pressure is the lowest circulatory pressure in an *elastic* circulatory system.	3. The intra-ocular pressure is the lowest circulatory pressure in a *rigid* circulatory system.
4. Variations in general circulatory pressure produce only physiological changes in the intra-ocular pressure.	4. Variations in general circulatory pressure produce, in consequence of 1 and 2, pathological results.

The Shallow Anterior Chamber in Glaucoma.

So far we have discussed Primary Glaucoma altogether as a question of raised vascular pressure within the unyielding sphere of the eyeball, and therefore we must now consider how under such conditions of vascular pressure the various clinical and pathological manifestations of Glaucoma are produced. The first of these sequelæ to be considered is the shallow Anterior Chamber existing in the acuter forms of Glaucoma, the cause of which it is easy to understand when we realize that, like the primary condition, it also results from the altered nature of the intra-ocular circulation.

Within the unyielding sphere of the eyeball the total volume of the solid and fluid contents is fixed. The tissue volume, with which may be included the viscid mass of the vitreous, is constant, so that the remaining space is occupied by the vascular volume of the blood and the extra-vascular volume of the aqueous. These two fluid volumes are complementary to each other, so that, if the one fluid volume increases or diminishes, this change in volume can only take place at the expense of the other fluid volume, and *vice versa.*

In Primary Glaucoma, as the circulation approximates that in a rigid system, the normal relative amount of blood in the arteries, capillaries, and veins, is altered. This vascular change, however, only takes place at the expense of the aqueous volume, with the result that the Anterior Chamber becomes shallow. If the aqueous were completely and absolutely shut off from all access to veins, then the aqueous volume would remain unchanged, just as the fluid contents of the "Glaucoma" flask (Fig. 39) remain throughout unaltered, because, unlike the aqueous, they are shut off completely from all access to the circulating fluid, whose volume must for the same reason also be constant. Thus it follows that as the pressure in the flask is raised the outflow tubing (at I) becomes compressed. This compression illustrates the alteration in the relative amount of fluid in the various parts of the tubing brought about in consequence of the conversion of its elastic walls into rigid ones, but it can only take place as the tubing at A expands.

Now, in the eye the extra-vascular aqueous is never absolutely shut off from contact with the Iris veins, and therefore at the outset of a glaucomatous attack, as the intra-ocular circulation tends towards a rigid system, the relative distribution of blood in its different areas becomes altered, and the total vascular volume in the eye is increased at the expense of the aqueous volume. In this fact lies the difference between the "Glaucoma" flask and the glaucomatous eye. In the former no diffusion can take place, and therefore the circulatory changes are brought about under a constant circulatory volume; but in the eye the obstruction is relative, and not absolute, and so diffusion takes place, by which the aqueous volume is reduced and the total circulatory volume increased.

The shallow Anterior Chamber in Glaucoma corresponds to and follows from the same causes which lead to the descent of the brain mass when the cerebral pressure is raised. The close anatomical and physiological points of resemblance existing between the intra-ocular and intra-cranial contents have already been discussed, and they naturally lead one to expect this pathological analogy. When, from whatever cause, the intra-cranial pressure is raised, the intra-cranial circulation tends to a rigid system, and the normal relationship of the volume of the blood in the arteries, capillaries, and veins, becomes altered.* To make room for the increased vascular volume, the extra-vascular volume of the cerebro-spinal fluid in the basal cisternæ is reduced, and the brain is driven downwards. Exactly the same process occurs in the eye in Glaucoma, as here likewise the total vascular volume is increased at the expense of the extra-vascular aqueous volume, thus producing the clinical manifestations of displacement forwards of the Lens and shallowing of the Anterior Chamber.

* Leonard Hill, *op. cit.*

SUMMARY.

1. The total volume of the solid and fluid contents of the eyeball is fixed.
2. Therefore the total vascular volume can only increase at the expense of the extra-vascular aqueous volume.
3. In Glaucoma, as the intra-ocular circulation approximates that of a rigid system, the relative amount of blood in the arteries, capillaries, and veins, is altered.
4. If aqueous were totally cut off from veins, then the total vascular and extra-vascular volumes would remain unchanged, as in the "Glaucoma" flask.
5. The obstruction being relative, and not absolute, the aqueous volume is displaced by increase of total vascular volume, and the Anterior Chamber becomes shallow.

Dilatation of the Pupil.

The dilatation of the pupil, like the shallowness of the Anterior Chamber, is also the outcome of the circulatory changes which culminate in the outbreak of a glaucomatous onset.

The circulation in the Iris is the most peripheral of the Uveal system, and therefore, when the pressure rises, the capillaries at the pupillary margin are the first compressed, and the pupil in consequence dilates. This obliteration of the Iris capillaries corresponds to the compression of the rubber tubing in the "Glaucoma" flask (Fig. 39). Being a vascular manifestation, the dilated pupil is frequently irregular, depending on the anatomical distribution of the vessels. As the pupil dilates, the Iris Crypts become closed, and thus the intra-ocular fluids are still further shut off from contact with the Iris Veins, so that the state of rigidity of the intra-ocular contents is still further augmented.

The dilatation of the pupil in Glaucoma is the converse of the condition present after Paracentesis, when the Iris becomes hyperæmic and the pupil contracted. Both these opposed conditions of the pupil are the direct result of alterations in the nature of the circulation, necessitating a readjustment of the individual volumes of the constituent fluid volumes that go to make up the total and fixed intraocular volume.

An instructive comparison may thus be drawn between the conditions formed after Paracentesis and those existing in Glaucoma, which may be tabulated as follow:

PARACENTESIS.	GLAUCOMA.
1. The eyeball a rigid but open sphere.	1. The eyeball a rigid and closed sphere.
2. The intra-ocular pressure below the normal intravenous level.	2. The intra-ocular pressure above the normal intravenous pressure.
3. Elastic vascular system with stasis.	3. Rigid circulatory system, with acceleration of blood-flow.
4. Aqueous volume primarily reduced; therefore total vascular volume increased and the anterior chamber shallowed.	4. Total vascular volume primarily increased; therefore aqueous volume diminished and anterior chamber shallowed.
5. Iris hyperæmic; therefore pupil contracted.	5. Compression of most peripheral pupillary vessels, and therefore pupil dilated.

Haziness of the Cornea.

The characteristic steamy appearance of the Cornea in Glaucoma is produced, as first pointed out by Fuchs,* by œdema of its epithelium and superficial lamellæ. This œdema, being limited to the superficial lamellæ of the cornea, cannot be due to filtration or passage of aqueous outwards under pressure, as in that case one would expect the posterior layers to be equally affected; which is not the case.

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* Fuchs, *Archiv für Ophth.*, vol. xxvii., 1882.

The explanation of this condition lies in the fact that normally the corneal tissue fluid is a diffusion, and not a filtrate, from the aqueous, and therefore, when the intra-ocular tension is raised, the tension in the corneal tissue is likewise raised to the same height throughout its whole thickness.

Under such conditions there is, on the surface of the cornea, a sharp and sudden drop of pressure to practically zero. This sudden difference between the raised tension of the corneal tissue fluid and the zero pressure of the atmosphere causes an œdema, which is naturally limited to the superficial strata of the cornea. The superficial œdema is, in fact, an expression of the disturbed relationship between the corneal tissue fluid pressure and the atmospheric pressure.

Normally, the difference between the corneal tissue tension and the atmospheric pressure is slight, but in glaucoma this difference becomes marked, and in proportion as it becomes accentuated does œdema appear on the corneal surface. The œdema is thus analogous to the condition produced on the surface of the skin by the application of an exhausted cupping-glass, which by removing the atmospheric pressure alters the relationship of the surface pressure to the tissue tension, thereby producing superficial œdema.

As already noted, the resistance which the fingers encounter when clinically testing the intra-ocular pressure is not made up of any single element, but is composed of different factors, of which the most important are the resistance and tension of the fibrous tunic of the eyeball.

As the aqueous diffuses through the unyielding corneoscleral envelope, the tissue fluid tension of the latter will fluctuate directly with the intra-ocular pressure, and therefore in Glaucoma, in proportion as the intra-ocular pressure is raised, the tissue fluid tension of the corneo-sclera is likewise raised to the same level, and in consequence its rigidity increased.

In the normal as in the Glaucomatous globe the intraocular volume is fixed, and therefore, on palpating the normal eye, we merely feel the normal intra-venous level of its contents pressure plus the normal tissue tension of the corneo-sclera ; in Glaucoma, on the other hand, the globe feels tense, because now we feel the raised intra-ocular pressure plus the raised tissue tension of the corneo-scleral envelope.

Occlusion of the Angle.

The occlusion of the Angle of the Anterior Chamber by the Iris Root has been regarded, since its discovery by Kneis and Webber, as one of the most important factors in bringing about an acute glaucomatous onset. This view, at first sight so attractive and plausible, is open to the very obvious objection that, if angular adhesion of the Iris Root were the important etiological factor it is supposed to be, there ought to exist some sort of relationship between the clinical manifestations and the degree and extent of occlusion found on pathological examination. I have, for instance, carefully examined the whole circumference of the Angle in two eyes removed for chronic Glaucoma, and found this region completely occluded by firmly adherent Iris. Had this occlusion been the true cause of the chronic and repeated attacks of Glaucoma, then these attacks, instead of being of the chronic congestive type, ought to have assumed the acutest possible form, and that long before time had been given to produce the total firm adhesion of the Iris which was present (Fig. 41). A consideration of the lack of correlation between this so-called cause and the effect that it is supposed to produce is alone sufficient to show that blocking of the Angle by the Iris cannot be the primary factor that it has been considered.

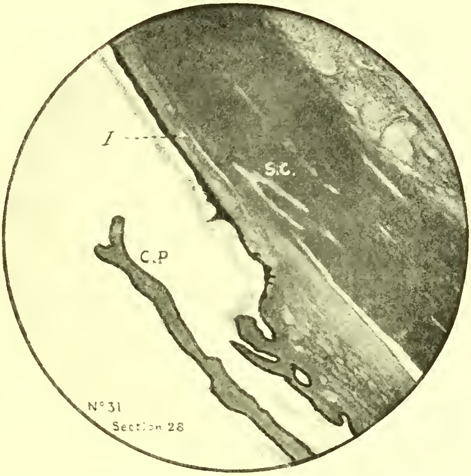

Fig. 41.—Micro-Photograph of Section of Chronic Glaucoma, from a Case of Mr. Richardson Cross.

Van Gieson's stain. Fixed in formalin. Schlemm's canal (S.C.), with efferent vessel. To the inner side of the sinus lies the sclerosed cribriform ligament. Adherent and atrophic iris (I.).

The atrophied and sclerosed ciliary process (C.P.) lies markedly displaced forward, and *not retracted.* The process appears in this section detached from the rest of the ciliary body, owing to unavoidable obliquity in sectioning. The adjoining sections show the attachment to the ciliary body, but then the most anterior point of this process is missed.

Glaucoma follows quite irrespective of the condition of occlusion of the Angle, a fact which is quite comprehensive when it is realized that the primary obstruction is produced by sclerosis of the Cribriform Ligament, and that in consequence of this the glaucomatous onset is determined by vascular conditions, whereby the circulation in the eye is converted from an elastic to a rigid system.

The effect of this alteration in the nature of the intra-ocular circulation is to give rise to a state of "congestive œdema" of the whole of the vascular intra-ocular tissues. This "congestive œdema" is an expression of tissue reaction, and it follows directly in proportion to the acuteness and degree of the glaucomatous onset. It can therefore be likened to the similar condition of the cerebral tissues when the intra-cranial pressure is raised.

In the case of the Iris, swelling and œdema of its tissue causes its base to become applied to the Cribriform Ligament, so producing blocking of the Angle.

This occlusion, far from being a cause of Glaucoma, is entirely a secondary pathological manifestation depending on the acuteness and degree of "congestive œdema" characterizing the whole of the intra-ocular tissues during a glaucomatous onset. The angular adhesion is, in fact, a mere localized and fortuitous sequel of a generalized tissue reaction, which, in consequence of the absence of room at the circumference of the Anterior Chamber, compels the surface of the swollen Iris Root to become applied to the back of the Cornea (Fig. 42).

"Congestive œdema" characterizes all acute and sudden changes in intra-ocular pressure, and hence blocking of the Angle is a constant and marked pathological feature in the acute Glaucomas; while in the chronic, non-congestive forms it is absent, and the Angle is found open. The blockage of the Angle is, in other words, a pathological manifestation of the degree of tissue reaction, and not one of etiological significance.

In the subacute or chronic-congestive forms of Glaucoma, the Iris, after the first congestive attack, may or may not form a permanent anterior peripheral attachment; but when such is once formed, each subsequent attack will cause more and more of the base of the swollen and congested Iris to move forward, and become first applied, and then adherent, to the posterior surface of the cornea.

As swelling and œdema occasion the application of the

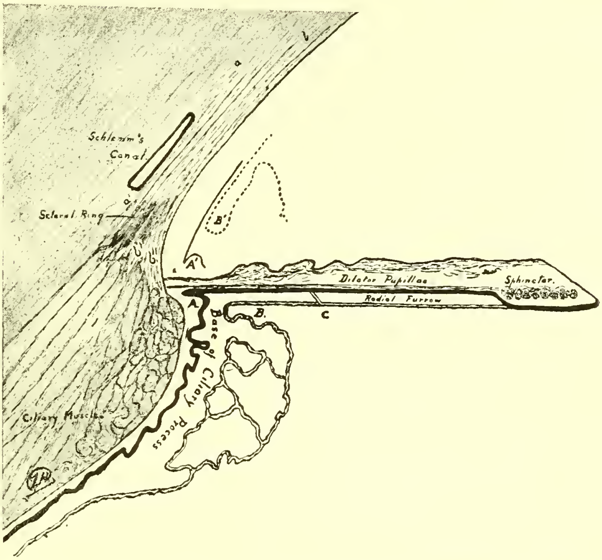

Fig. 42.—Diagram of Iris and Ciliary Body, reconstructed from Serial Sections of Normal Eye, and illustrating in Thick Black Line an Inter-Ciliary Groove, and its Continuation forward at A into an Iris Radial Furrow (*cp.* Fig. 19).

The ciliary process arises at B from a base which is a continuation of the iris stroma. A section which starts peripherally along a radial furrow at A, and then jumps out of it as at C, will give the appearance as if the iris root had been compressed by the ciliary processes (see Fig. 20).

As the iris swells in consequence of congestion and œdema, its anterior surface at its attachment becomes, for lack of room, applied to the inner aspect of the cribriform ligament, and finally to the posterior surface of the cornea.

The ciliary processes, in consequence of the connection between their bases and the iris root, become rotated forward, so that the points A and B come to lie at A′ and B′, and, once the iris has become adherent, will remain permanently anchored in this position (Fig. 41).

Iris to the back of the cornea, it follows that the anatomical distribution of the vessels at the Iris periphery plays an important part in determining the state and extent of the anterior peripheral synechia of the Iris, accounting for the not infrequent pathological finding of an extensive adhesion on one side of the circumference of the Anterior Chamber, and an open or only partially occluded Angle on the other.

The Position of the Ciliary Processes in Glaucoma.

The peripheral adhesion of the Iris circumference is always associated with forward displacement of the Ciliary Processes (Fig. 41). This position of the Ciliary Processes is met with, not merely in the congestive, but also in the atrophic stage of Glaucoma; in other words, it is a constant accompaniment of the pathological manifestation of blocking of the Angle by the Iris quite irrespective of recent congestion.

According to Priestley Smith, it is the Ciliary Processes that push the Iris into the Angle, and therefore on the subsidence of swelling the Processes retract.

Now, if the Iris Root were pushed forward by the congested Ciliary Processes, then once the congestion is relieved, and still more so when atrophy is taking place, the Ciliary Processes ought certainly to spring back to their original position. Serial sections show, however, that, in every case in which the Iris is adherent, the Ciliary Processes, whether atrophied or not, are invariably found in a forward position, and not retracted (Fig. 41). In cases in which the Angle is occluded on one side, but open on the other, serial sections show that the Ciliary Processes on the side where the Angle is open are atrophied in their ordinary position, while on the other side they are likewise atrophied, but displaced forwards. The appearance in a random section of a retracted and atrophied Ciliary Process with an adherent Iris is due to the section only showing part of the Process, and not going completely through it; hence it is absolutely necessary that serial sections be studied, as only in this way can the part be reconstructed as it actually existed in the living eye. In viewing random sections of specimens in which the Ciliary Processes are atrophied, it must not be forgotten that the inter-ciliary spaces are proportionately larger, and therefore the number of sections going through a single Ciliary Process are fewer than normal, while, *per contra*, the number going through the inter-ciliary spaces are much more numerous.

A consideration of the facts demonstrated by serial reconstruction shows that it cannot be the Iris which is pushed forwards into the Angle by the Ciliary Process, but that, on the other hand, it is the Ciliary Processes which are dragged forward by the Iris after congestive œdema has caused its root to become applied, and then adherent, to the back of the cornea.

The Ciliary Processes arise, as already pointed out, from a base which is continued forward into the Root of the Iris (Fig. 42). Now, when the surface of the Iris Root becomes adherent to the inner aspect of the Cribriform Ligament, it is obvious that the base of the Ciliary Processes must be pulled upon, so that the points A and B come to occupy the position of A′ and B′, with the result that the Ciliary Processes become anchored in a forward position quite irrespective of either subsequent œdema or atrophy.

In acute Glaucoma, Priestley Smith describes and illustrates moulding of the Ciliary Processes into a wedgelike form by compression between lens and Iris.*

The real explanation of this appearance is, however, clear when serial sections are studied. The " moulding " is simply the result of the difficulty of getting sections to pass along the whole length of a Ciliary Process, and thus, while the anterior part of a Ciliary Process will be shown in one section, its posterior part will be left out, and only seen in the adjoining sections. Random sections cannot illustrate the true state of affairs; they can only show all sorts of variations in the associations between the apices and bases of the Processes. Serial reconstruction is, in fact, the only method by which the true relation of the Iris Root to the Ciliary Processes can be made out in either normal or pathological conditions, and it demonstrates that such a condition as "moulding" of the Ciliary Processes does not exist.

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* Priestley Smith, "Glaucoma," 1891; also article in Norris and Oliver, " System of Ophth.," vol. iii., p. 647, fig. 22.

The appearance of so-called "compression" of the Iris Root in cases of Glaucoma with an open Angle is but another result of incomplete examination. This manifestation has already been discussed, and shown to be produced by the presence of the Iris Radial Furrows. If a section is cut so as to include the outer portion of an Iris Radial Furrow (from B to C, Fig. 42), the posterior Iris surface will appear notched; but this appearance is not due to Compression of the Iris root by the Ciliary Processes, but is merely the result of the posterior Iris surface being a plicated, and not a smooth surface. This notching will therefore be met with in sections of normal eyes (Fig. 20), as well as of cases of Glaucoma in which the Iris is non-adherent, but only in such sections as pass along the outer part of an Iris Radial Furrow.

When the Iris Root is adherent to the corneal surface, that portion of it so attached is the part which manifests the earliest and most marked signs of atrophy (Fig. 42). This atrophy of the adherent portion of the Iris is the direct result of the increased hydrostatic pressure within the eye, acting solely through the posterior surface of the Iris, and so crushing the Iris stroma against the rigid cornea. In the free, non-adherent portion of the Iris, the hydrostatic pressure acting on the Iris tissue is the same on both its surfaces, and therefore atrophy, however marked it may be, is never so advanced as at the spot where the Iris is adherent. The notching here is an atrophy existing throughout the whole circumference of the attached Iris, and therefore quite a different manifestation from the appearance caused by the Iris Radial Furrows when the Angle is open. It is essential to differentiate clearly between these two forms of notching which may be encountered at the Iris Root. The one seen when the Angle is open has no relationship whatever to the rise in the intra-ocular pressure, while the other is the direct outcome of the uneven incidence of a raised hydrostatic pressure on the adherent Iris tissue. In neither case do the Ciliary Processes play the part of compressing agents.

Fallacies in Priestley Smith's Theory.

Priestley Smith holds that the predisposing factor in Primary Glaucoma is the encroachment of the growing Lens on the circumlental space, whereby congestion of the Ciliary Processes will lead to occlusion of this area, and check the flow of surplus fluid from the Vitreous. The retained volume behind the Lens causes this latter to push forward and press the Ciliary Processes against the Iris Root, and so produce blocking of the Angle. As regards this mechanism, it is unnecessary to discuss it again, as the anatomical evidence on which it is based has already been shown to be inadequate. Here the experimental evidence only will be discussed.

The essential experimental basis on which Priestley Smith's theory rests is the possibility of inducing a pressure discontinuity between the Aqueous and Vitreous Chambers, whereby the lens is driven forwards, the Anterior Chamber shallowed, and the Angle occluded. The existence of such a pressure discontinuity Priestley Smith considers proved by his filtration experiments on excised sheep's eyes, in which a hollow needle, connected by a length of rubber tubing with a reservoir, is inserted into the Anterior Chamber, and another similarly connected into the Vitreous Chamber (see Fig. 44). The reservoirs are at first both raised to the same level above the eye, with the result that "the pressures in the Aqueous and Vitreous Chambers are equal; the Lens and Iris are not displaced; the filtration angle remains open; the fluid quickly injects the episcleral veins and flows from their cut ends."*

It is essential to note, as has been already pointed out in discussing the nature of the aqueous outflow, that an experiment such as this is in no way comparable to the conditions existing in the living eye, in which the blood-circulation is active, and in which aqueous and venous pressures stand at the same level. The coloured fluid that injects the episcleral veins is truly a filtrate, because there is no circulation and the veins are empty, and therefore under such artificial conditions no deductions can be made as to aqueous resorption in the living eye.

If the level of the reservoir connected with the Vitreous Chamber is raised above that connected with the Anterior Chamber, Priestley Smith points out that "the vitreous pressure is a little higher than the aqueous pressure; the Lens and Iris advance; the Anterior Chamber is shallow; the filtration angle is closed; there is no injection of the episcleral veins, and no escape of the coloured fluid at any part of the eye, even after several hours." As far as they go, these observations are in themselves perfectly correct, but again it is altogether faulty analogy to compare the conditions and findings here to the conditions existing in the closed sphere of the eye. In this experiment the Anterior Chamber is not a closed space, but an open one connected with an open reservoir, and the conditions in such an open Chamber are in no way comparable with those existing in the closed corneo-scleral envelope, in which the pressures in the Aqueous and Vitreous Chambers are always the same, and move together under all varying hydrostatic pressures. This fact admits of very simple proof (Fig. 43). A Leonard Hill's manometer gauge is connected by a hollow needle with the Anterior Chamber of an excised sheep's eye, while a similar needle, connected by rubber tubing with a pressure bottle, is inserted into the Vitreous Chamber. As the pressure in the Vitreous Chamber

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* Priestley Smith, "Glaucoma," 1891; also in Norris and Oliver, "System of Ophth.," 1900, vol. iii., p. 632.

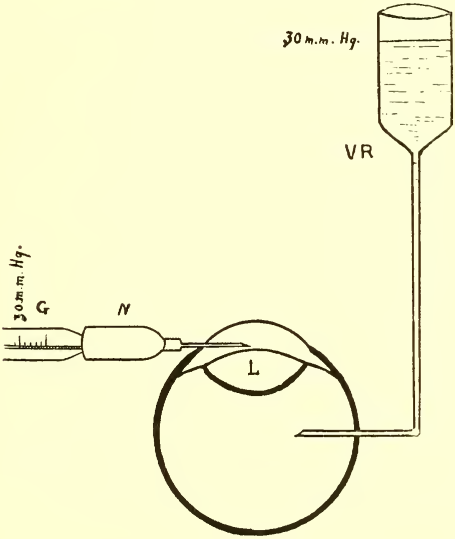

Fig. 43.—Measurement of Variations in Vitreous Pressure in Excised Sheep's Eye.

Pressure bottle (V R) connected by rubber tubing, with hollow needle inserted into Vitreous Chamber.

The pressure in the Anterior Chamber is registered by a Leonard Hill's gauge (G), which is connected with a hollow needle by a glass nozzle (N).

Every variation in height of the pressure bottle (V R) is immediately registered by the gauge in the Anterior Chamber, proving that the pressure on either side of the Lenticulo-zonular diaphragm (L) is always the same from zero upwards.

is varied by raising or lowering the pressure bottle (V R) the pressure registered by the manometer (G) in the Anterior Chamber varies absolutely and directly with the vitreous pressure.

The manometer gauge being registered to millimetres of mercury, a simple calculation is necessary to convert the height of the pressure of the column of water of the vitreous reservoir (V R) into millimetres of mercury (13·5 centimetres $H_2O = 10$ millimetres Hg). If the pressure bottle be raised 33·75 centimetres above the eye, then the manometer gauge in the Anterior Chamber registers the same pressure only in millimetres of mercury—*i.e.*, 25 millimetres Hg. If the pressure bottle is lowered, say, to 27 centimetres, the manometer scale drops to 20 millimetres Hg, and so on.

So long as the Anterior Chamber remains a closed space, every variation in Vitreous pressure produces a corresponding alteration in the Aqueous pressure, so that the two pressures always vary together the same amount. As one continues alternately raising and lowering the pressure bottle, and so varying the intra-ocular pressure, one finds that in time the Anterior Chamber empties and the Lens and Iris come forward; while the gauge still registers the same pressure as that in the vitreous chamber, so long as any fluid is left in the Anterior Chamber. The cause of the advancement of the lens does not lie, however, in any difference in pressure between the two chambers, but is simply due to the aqueous filtering out of the Anterior Chamber under the hydrostatic pressure transmitted to it from the Vitreous.

As the Aqueous filters away, its place in the intra-ocular volume is taken by fluid from the "vitreous" pressure bottle entering the Vitreous Chamber, and therefore, as the Aqueous drains away, the Lens moves forward. The Anterior Chamber thus becomes shallow, not from a difference in pressure, but from a diminishing volume, the pressure always remaining the same as that in the Vitreous Chamber.

Manometric observations show that, contrary to Priestley Smith's theory, so long as the Anterior Chamber remains a closed space, the balance of pressure between it and the Vitreous Chamber is perfect; so that if the vitreous pressure is raised, the aqueous pressure mounts to exactly the same height, while if the pressure is lowered the same equality exists.

The fallacy in Priestley Smith's deductions from his pressure experiments lies in regarding the Anterior Chamber as a closed space, when as a matter of fact, by its connection with a reservoir (A.R.), it is an open one (Fig. 44). In consequence of this condition the transmission of hydrostatic pressure in the Anterior Chamber is fundamentally altered, as can be easily demonstrated by repeating Priestley Smith's experiments with a Leonard Hill's gauge (G) connected with the Anterior Chamber, at the same time placing a tap (T) in the course of the tubing leading from the Aqueous Reservoir (A.R.), so that the Anterior Chamber can be readily converted from an open to a closed chamber, and *vice versa*, by a simple turn of the tap.

If the tap is closed, the fluid in the "Aqueous" reservoir is shut off, and the conditions present are exactly the same as in the experiment already described (Fig. 43); for the pressure registered by the gauge moves with the height of the column of water in the "Vitreous" Reservoir (V.R.).

If now the two pressure bottles are placed at the same level, and the tap (T) is opened, the gauge (G) registers in millimetres of mercury the pressure of the column of water at which the reservoirs stand—say 25 millimetres Hg. The pressure in both Aqueous and Vitreous Chambers is the same, but if the pressure bottle (V.R.) connected with the Vitreous Chamber is raised above the level of the Aqueous reservoir (A.R.) by 6·75 centimetres, which corresponds to a rise of 5 millimetres Hg, we find that the gauge in the Anterior Chamber still registers only 25 millimetres Hg, while the pressure in the Vitreous Chamber will be 30 millimetres Hg. The conditions are, of course, exactly as Priestley Smith has quite correctly stated, that the pressure in the Vitreous Chamber is higher than that in the Aqueous; but where he is altogether wrong is in regarding the Anterior Chamber here as the same as in the closed sphere of the eyeball. No such comparison can be made;

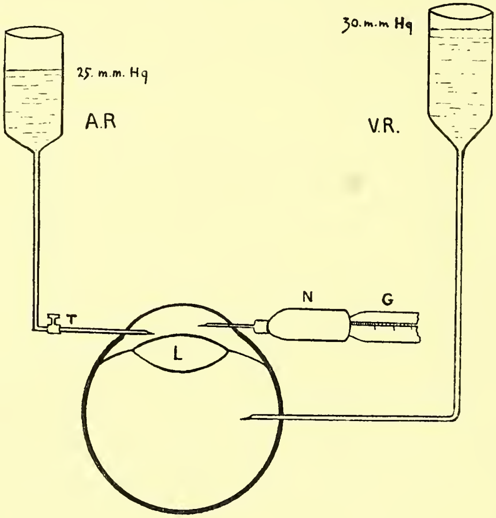

Fig. 44.—Priestley Smith's Pressure Experiment.

Pressure bottles (A.R. and V.R.) are connected respectively with Aqueous and Vitreous Chambers. Leonard Hill Gauge (G) connected with Anterior Chamber. The tubing leading from the Aqueous Reservoir (A.R.) is supplied with a tap (T); if this is closed, the conditions are exactly the same as in Fig. 43, and the pressure registered varies with the level of the Vitreous Reservoir (V.R.).

If the tap is open, the Anterior Chamber is not a closed space, and the pressure registered will always correspond to the level of the Aqueous Reservoir (A.R.); but as soon as the tap (T) is closed, the pressure mounts again to the level of the Vitreous Reservoir (V.R.).

for if now the tap (T) is shut, the Anterior Chamber at once becomes a closed space, and the pressure registered by the gauge immediately mounts to 30 millimetres Hg—*i.e.*, the Vitreous pressure; while as soon as the tap is opened again, the pressure in the Anterior Chamber falls again to 25 millimetres Hg.

The cause of this variation in pressure in the Anterior Chamber lies in the fact that when the tap is shut the Anterior Chamber is a closed space, and therefore the pressure throughout the intra-ocular contents is a uniform one derived from the height of the "Vitreous" pressure bottle; but as soon as the tap is opened the aqueous chamber is no more a closed space, but an open one, and the pressure within it must correspond to the column of fluid in the "Aqueous" pressure bottle. The result is simply what one would expect from the well-known hydrostatic law that water finds its own level. If there were free communication between the two chambers, the fluid in the pressure bottles would simply flow from the higher to the lower one, and the pressure in the eye would represent the mean. In consequence, however, of the presence of the Lenticulo-zonular diaphragm (L), such a flow can only take place to the extent of driving all the fluid out of the Anterior Chamber into the "Aqueous" Reservoir, and therefore the pressure in front of the Lens can only represent the height of the "Aqueous" pressure bottle. As the Anterior Chamber is open to the "Aqueous" Reservoir, the pressure in it can never under such circumstances rise above this level, but immediately the tap is turned, and the Anterior Chamber made a closed space, the pressure in front and behind the Lens becomes equalized to the higher level of the "Vitreous" pressure.

It is instructive to note that we can reverse the experiment—that is, raise the pressure in the Aqueous Chamber above that in the Vitreous Chamber, with the same results as regards the pressure registered by the gauge in the Anterior Chamber, depending on whether the tap is open or closed.

If we raise the Aqueous pressure to correspond to 30 millimetres Hg in the manometer scale, and drop the Vitreous pressure to 25 millimetres Hg, then, so long as the tap is open, the manometer in the Anterior Chamber will register 30 millimetres Hg. The pressure in the Anterior Chamber is higher than that in the Vitreous Chamber. When the tap is shut, making the Anterior Chamber a closed space, the pressure in it drops down to the Vitreous level of 25 millimetres Hg, though this drop in pressure may take some slight appreciable time to register, owing to the viscosity of the Vitreous. When the tap is again opened, the pressure registered in the Anterior Chamber at once rises to 30 millimetres Hg.

These pressure experiments, as so conducted, demonstrate in the most striking manner that in the closed eyeball the pressures in Aqueous and Vitreous Chambers are always the same, the delicacy of the balance being shown in the ready response by which a slight difference in level of only a few millimetres of mercury between the two chambers is at once equalized when the Anterior Chamber is restored to a closed space. Incidentally the experiments also prove the accuracy of the manometer gauge, which registers with equal trustworthiness the slightest alterations in pressure.

When we thus analyze the experimental findings obtained by Priestley Smith, on which so much of his theory of Glaucoma depends, we find that, far from supporting his hypothesis that in Glaucoma the Vitreous pressure becomes greater than the Aqueous pressure, they prove that no such dissociation of pressure can exist so long as the eyeball remains a closed sphere.

It is certainly true that experimentally the pressure of the Vitreous can be raised above that of the Aqueous, but it is equally true that such a condition finds no parallel when the Anterior Chamber is a closed space. In the closed sphere of the eyeball, whatever the level of the intra-ocular pressure may be, it is identical in both Aqueous and Vitreous Chambers. Apart, therefore, from all other considerations of the theory which Priestley Smith has advanced to account for Primary Glaucoma, that part of it which assumes a difference in pressure between Vitreous and Aqueous is erroneous.

Summary of Contra-Indications to Priestley Smith's Theory.

1. The pressure in Glaucoma is not a question of volume, but of circulatory pressure, the total volume being constant.
2. In the closed sphere of the eyeball the pressures in Aqueous and Vitreous Chambers are always evenly balanced.
3. The advancement of the Lens and the shallow Anterior Chamber are the result of displacement of aqueous volume by the increased vascular volume produced by the circulation running in rigid lines.
4. Reaction of the intra-ocular tissues to the altered conditions of the circulation causes "congestive œdema," resulting in swelling of the Iris and application of its Root to the Cornea.
5. Displacement of the Iris Root causes traction on the bases of the Ciliary Processes, which thus become anchored in a forward position irrespective of subsequent atrophic changes.
6. This position of the Ciliary Processes is an effect, and not the cause of the Iris adhesion.
7. The adherent area of the Iris circumference is the first part of its stroma to manifest atrophic changes, as the raised hydrostatic pressure acts only on the posterior surface, so crushing the stroma against the rigid Cornea.
8. The notching of the Iris Root seen in normal eyes, or in Glaucoma with open angle, is an artificial appearance produced by the presence of the Iris Radial Furrows.

The Clinical Types of Primary Glaucoma.

Primary Glaucoma manifests itself in distinct types, which are not, however, hard-and-fast forms of the disease, but mere expressions of differences in degree of reaction to the same primary forces.

The constant pathological factor in Primary Glaucoma is sclerosis of the Cribriform Ligament, while the characteristic features which distinguish the two extreme clinical types of Acute Congestive and Chronic Non-congestive Glaucoma are the result of, and are produced by, conditions directly affecting the intra-ocular pressure through the general circulatory pressure. It is a question of degree of action of the vascular factor, not a difference in the nature of the causes.

The sudden and rapid onset of increase in general vascular pressure produces an equally sudden rise in the intra-ocular pressure, which in an eye with diminished access of its fluid contents to veins will result in the rapid conversion of the elastic intra-ocular circulation into a rigid one. The effect of such a sudden change in the circulation is to disturb the normal conditions of cellular life of the tissues, and so call forth an acute reaction on their part, which finds expression in "congestive œdema," and as a final result in atrophy.

The primary factors in chronic, simple, or non-congestive Glaucoma are exactly the same as those that determine an acute attack, only differing in degree. The pathological changes are brought about more slowly, and secondary alterations have time to become established, so that, instead of acute reaction on the part of the tissue cells, the manifestations produced are those of chronic reaction in the form of fibrosis or atrophy. The clinical and pathological features are thus altered, although the exciting mechanical causes are exactly the same.

The characteristic clinical and pathological feature of Chronic Glaucoma is atrophy of the tissues, associated with the multiplication of venous channels. Both these manifestations are an expression of the chronicity of the condition.

In a rigid system of tubes the greatest resistance is encountered at the outflow, and the same applies to the intra-venous pressure when it is raised. In consequence, when the condition is chronic, new vessels are formed in the attempt to overcome the resistance at the venous exit. The intercommunicating branches of the Anterior Uveal Veins in the Sclera thus become vastly increased in number and size, and the same applies to the Iris Veins.

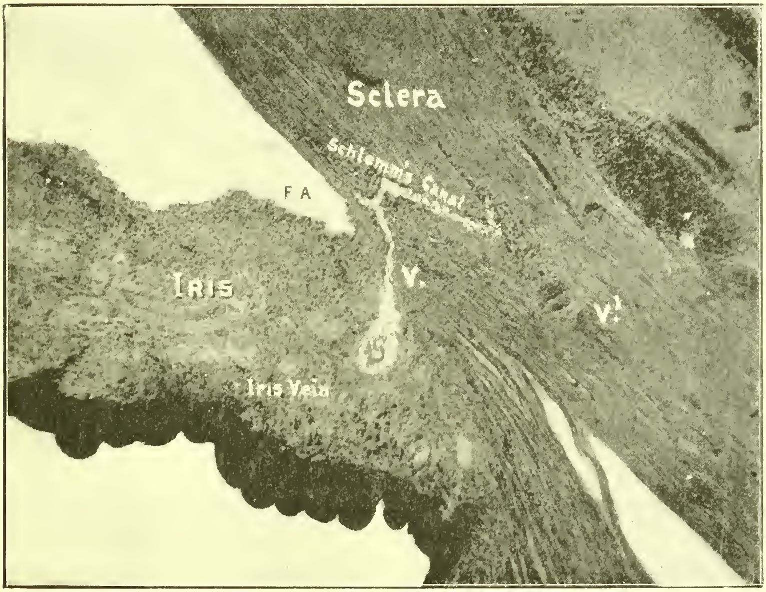

Fig. 45.—Chronic Glaucoma.

Micro-photograph showing connecting Vein (V.) between an Iris Vein and Schlemm's Canal. The vessel contains blood-corpuscles.

The Angle of Anterior Chamber (F.A.) not altogether occluded.

In cases of Chronic Glaucoma with occlusion of the angle, I have very frequently found, as the result of serial sectioning, new-formed and direct tributaries connecting the Iris Veins with Schlemm's Canal. Under normal conditions the association between the Iris Veins and Schlemm's Canal is always an indirect one, but in Chronic Glaucoma with partial (Fig. 45) or total occlusion (Fig. 46) direct communications are formed through the sclerosed Cribriform Ligament. These connections exemplify the important rôle that the venous system must play under both physiological and pathological conditions.

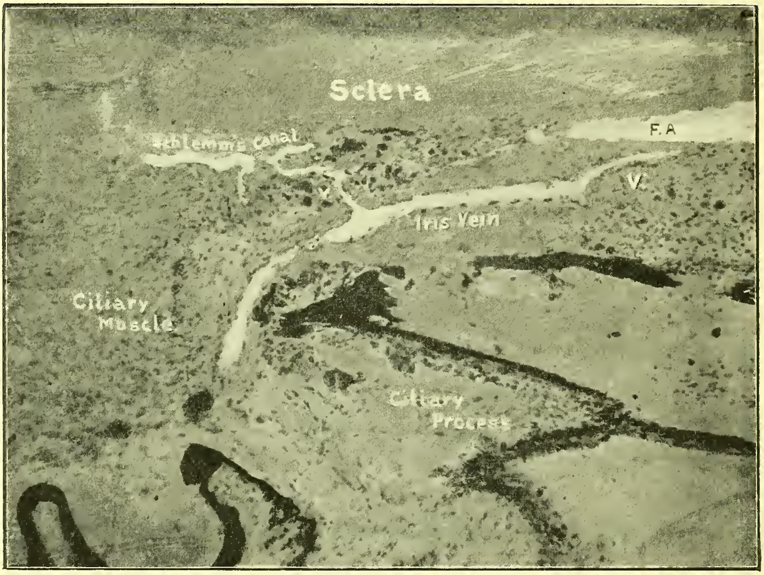

Fig. 46.—Direct Iris Tributary to Schlemm's Canal in Chronic Glaucoma.

The Iris Root is adherent to the whole length of the Cribriform Ligament. False angle of the Anterior Chamber (F.A.).

Schlemm's Canal is connected by a vessel (V.) with an Iris Vein, which latter is joined by a new-formed superficial Vein (V.').

Secondary Glaucoma.

The numerous clinical combinations which may lead to Secondary Glaucoma can only be harmoniously correlated with the Primary Disease when the vascular nature of the normal intra-ocular pressure is grasped. This physiological consideration lies at the very root of all forms of Glaucoma, and forms the common bond which links together all the various clinical manifestations of raised intra-ocular pressure, whether primarily or secondarily induced.

In Primary Glaucoma the predisposing and exciting factors are always constant, though varying in degree and intensity; but in Secondary Glaucoma they are multiple, and therefore each case must be accounted for individually, depending, as it does, on the pathological condition previously existing in the eye.

Although multiple in origin, the principles involved in the nature of the factors in Secondary Glaucoma are essentially the same as in the Primary Disease. The pathological pressure must be derived from the circulatory force, and therefore in all cases of Secondary Glaucoma the ultimate source of the rise in pressure can be traced directly to two causes—either a rise in intra-ocular circulatory pressure or diminished access of aqueous to veins, conditions which together constitute the essential factors of Primary Glaucoma. Under such circumstances, it would be out of place here to go in detail through the considerable list of intra-ocular conditions which are liable to set up glaucomatous tension, and therefore I will only refer briefly to a few varieties which illustrate the genera principles under which Secondary Glaucoma may become manifest.

From an etiological standpoint we can divide Secondary Glaucoma into two classes—a vascular and an obstructive—each of which is correlated with the corresponding factor of Primary Glaucoma. The *vascular class* of Secondary Glaucoma arises as the result of increased intra-ocular circulatory pressure at the outset, limited to a localized vascular area within the eye. Under this class come cases of increased pressure due to Cyclitis, Intra-ocular Tumour, Panophthalmitis, etc.

Here the contact between the intra-ocular fluids and veins is unimpeded ; but the vascular pressure is raised, and therefore, however free the contact may be, if the intra-venous pressure is raised, the intra-ocular pressure must rise to the same level, because such must always remain the lowest hydrostatic pressure within the closed sphere of the eyeball. The local causes which produce this type of Glaucoma all act through a raised intra-ocular circulatory pressure, however induced, and remain effective so long as the high circulatory pressure lasts, and the eyeball remains a closed sphere. When the intra-venous pressure falls to normal limits, the intra-ocular pressure follows suit, while if the eyeball is opened the pressure within is relieved, because hydrostatic pressure cannot be maintained in an open receptacle.

In considering Secondary Glaucoma, it is important to bear in mind the nature of Schlemm's Canal and the close intercommunication between the Anterior and Posterior Uveal Venous Systems.

In consequence of this close association, a rise of vascular pressure in one localized venous area, caused by inflammation or tumour formation, will readily spread to, and produce a rise of pressure within the venous sinus of Schlemm's Canal, and thereby raise the intra-ocular pressure. The Secondary Glaucoma so produced is analogous to the raised intra-cranial pressure accompanying Meningitis, Cerebral Tumour, or Abscess Formation.

The circulatory conditions that induce this type of Glaucoma are essentially intra-ocular in inception, in contradistinction to those that obtain in Primary Glaucoma, where the circulatory disturbances are generally induced by and associated with the general circulatory pressure, on which the physiological variations of the intra-ocular pressure normally depend.

In the *Obstructive class* of Secondary Glaucoma the increased pressure is brought about by a diminished access of aqueous to veins, such as by *occlusio* or *seclusio pupillæ*, etc.

The conditions here are materially different from those in the preceding class, as it is not the circulation which is primarily at fault, but the *fons et origo mali* lie in conditions which preclude the access of aqueous to veins.

If the aqueous is debarred direct access to veins, the intra-ocular fluid volume acts as a rigid, incompressible mass which transforms the nature of the circulatory flow from an elastic to a rigid system, thereby raising the venous or outflow pressure towards the arterial or inflow pressure. It is this conversion of the circulation into a rigid system that raises the intra-ocular pressure in the obstructive types of Glaucoma, by the same mechanism as that by which the pressure in the "Glaucoma" flask (Fig. 39) is raised.

Glaucoma in Serous Cyclitis or Iritis.

The raised intra-ocular pressure accompanying Cyclitis is essentially an example of the class of Secondary Glaucoma induced by a localized rise in vascular pressure. The altered inflammatory tissue state of the Ciliary Body leads to a raised pressure of its venous return, and this, in consequence of the close association between the Anterior Uveal Veins and the venous sinus of Schlemm's Canal (Figs. 23 and 24), will lead to a rise of pressure within the latter, and therefore to the aqueous pressure rising to the same level. Increase in aqueous pressure produces, as already experimentally shown, a corresponding rise in vitreous pressure, the consequence being that the Posterior Uveal or Choroidal Veins are pressed upon by the raised vitreous —*i.e.*, intra-ocular—pressure, and that their intra-venous pressure is raised to the same level.

As the venous volume in the choroidal plexus diminishes under the increased intra-ocular pressure, the aqueous volume increases and the Anterior Chamber deepens. In other words, the increased vascular volume of the Ciliary Body in Cyclitis, and the excess of aqueous present, can only exist within the circumscribed corneo-scleral envelope at the expense of the venous volume in the choroidal plexus, while the intra-ocular pressure is raised because of the increased intra-venous pressure in Schlemm's Canal.

Hypersecretion is only a feature of secondary importance in the sequence of events produced by inflammatory conditions of the Ciliary Body, the essential factor being the influence that the localized inflammation has in raising the intra-venous pressure of Schlemm's Canal.

Hypersecretion by itself cannot cause increase of intra-ocular pressure so long as the pressure within Schlemm's Canal stands at the normal intra-venous level, for the aqueous could never be contained in the Anterior Chamber at a higher pressure than that within Schlemm's Canal. When, however, the pressure in Schlemm's Canal is raised, the aqueous pressure—that is, the intra-ocular pressure—must rise *pari passu* with every increased millimetre of pressure within Schlemm's Canal. At the outset of Cyclitis the passage of aqueous to veins is free and uninterrupted, but in the later stages deposition and organization of inflammatory products in the meshes of the Cribriform Ligament may take place, causing an obstruction to the passage of aqueous to Schlemm's Canal which will lead to a sequence of events similar to those in Primary Glaucoma. At the outset of Cyclitis, however, such an obstruction cannot be effective either in inducing the raised pressure or in leading to accumulation of Aqueous; for if such were the case, the condition of the circulation in the eye would be at once reduced to a rigid system, as in the Primary disease. But, further, if obstruction existed, then Atropine, by closing the Iris Crypts, and thereby still further diminishing the range of access of aqueous to Veins, would always make matters infinitely worse.

The beneficial effects of Atropine follow from its influence in reducing the primary inflammatory condition of the Ciliary Body, and thereby diminishing the Intra-ocular pressure by lowering the venous pressure in the Anterior Uveal Veins, and therefore in Schlemm's Canal.

In Cyclitis the access of aqueous to Schlemm's Canal and to the Iris Veins is free and sufficient, but the pressure in the veins themselves is raised, and therefore the intra-ocular pressure has no option, but must follow the rise in the intravenous pressure determined by the inflammatory condition in the eye.

Glaucoma with Intra-Ocular Tumours.

The conditions which cause an intra-ocular tumour to give rise to increased pressure are essentially of the same nature as those which induce a growth in the analogous closed cavity of the cranium to set up increased intracranial pressure. In neither case is it a question of increased volume, but of alteration in the nature of the circulatory flow; for a growing tumour mass, whatever its position, will always accommodate its increasing tissue volume at the expense of the rest of the contents volume.

Physiologically increased tissue activity determines increased blood-supply to a part, and therefore increased circulatory pressure within it. If such is the case, then, if the circulatory pressure within a growing intra-ocular tumour is raised, this pressure will be transmitted to the adjacent and linked vascular areas.

That the circulatory pressure in an actively growing tumour is raised is demonstrated by the formation of a subretinal serous exudate, which could not be formed unless the vascular pressure in and about the tumour were above that of the rest of the Choroid.

In a Choroidal Sarcoma the initial localized rise of pressure is in the Posterior Uveal Vascular System, and therefore the converse of the condition met with in Cyclitis, where the Anterior Uveal Vessels are primarily affected. So long as the intra-venous pressure within Schlemm's Canal remains uninfluenced, the intra-ocular pressure will remain normal, no matter how large the growth may be, for the intra-ocular pressure will always lie at the lowest circulatory level.

As soon, however, as this lowest pressure is raised, Glaucoma will be produced. Thus it is that a tumour in the Ciliary Body will produce Glaucoma earlier than one farther back, as the vascular pressure in Schlemm's Canal will be the earlier affected. When the pressure in the Anterior Uveal Veins is raised simultaneously with a rise in the Posterior Uveal Veins, it means that the whole, and not merely a part, of the intra-ocular circulation is tending to a rigid system, which is exactly what occurs in Primary Glaucoma, in which, although the primary mechanism is somewhat different, the mechanical results and clinical manifestations are similar.

Glaucoma induced by Mydriatics.

Glaucoma following the use of mydriatics, after the presbyopic age has been reached, is an example of the obstructive class of Secondary Glaucoma, as it is directly induced by the diminished access of aqueous to veins. The accepted textbook explanation ascribes the result to the dilatation of the pupil, causing the Iris Root to encroach upon and obliterate the Angle of the Anterior Chamber. This theory, at first sight so simple and rational, is, after all, only explaining *ignotum per ignotius;* for if such were the true facts of the case, we should still have to account for the immunity from all such risks in the earlier years of life. The shallow Anterior Chamber of old age cannot be considered as a predisposing factor; for if it were, the danger of inducing Glaucoma would be equally great, if not greater, in the infantile eye, with its very shallow chamber.

In a presbyopic eye the route to Schlemm's Canal is impeded by the physiological sclerosis of the Cribriform Ligament, and therefore dilatation of the pupil, by closing the Iris Crypts, shuts off the Aqueous from the Iris Veins. We have here duplicated the same mechanical conditions as in the "Glaucoma" flask (Fig. 39). The intra-ocular fluids are shut off from contact with veins, and therefore the circulation in the eye is forced into rigid lines.

The pathological manifestations produced by dilatation of the pupil in the presbyopic eye will depend on the extent of the sclerosis of the Cribriform Ligament and therefore on the pre-existing obstruction of aqueous to Schlemm's Canal. If the passage to Schlemm's Canal is sufficiently free, it is immaterial whether the Iris Crypts are open or closed, but it is otherwise when sclerosis has already reduced this exit. Mydriatics will then readily induce Glaucoma.

Myotics, by their contrary effect on the pupil and Iris Crypts, are proportionately beneficial by facilitating the contact between aqueous and the Iris Veins.

Glaucoma following Traumatic Cataract.

The imbibition of aqueous by the lens fibres causes the latter to swell up and occupy the volume of the aqueous which in consequence is removed. In the absence of aqueous, the uveal circulation, being contained between the unyielding sclera and the viscous and incompressible masses of the vitreous and lens matter, becomes converted into a rigid system, with all its evil consequences.

A very similar condition is produced by the dislocation *en masse* of the lens into the anterior chamber, which, by displacing the fluid in that chamber, prevents the ready access of aqueous to Anterior Uveal Veins, whereby the intra-ocular circulation is forced along rigid lines.

Once more I would emphasize the fact that the raised pressure in the " obstructive " type of Secondary Glaucoma does not follow the retention of intra-ocular volume, but is induced by all such conditions as debar the aqueous from contact with the venous circulation, whereby the fluids contained in the eye act as a rigid volume. The hydraulic principles involved are, in fact, the same as those in the " Glaucoma " flask (Fig. 39).

SUMMARY OF THE ESSENTIAL FACTORS IN SECONDARY GLAUCOMA.

1. In Secondary Glaucoma, as in the Primary disease, the raised pressure is derived from the circulatory pressure, and is not the product of retained volume.
2. The immediate causes may be classified into—

   (*a*) Vascular, when a localized (inflammatory) rise in vascular pressure in the eye ultimately effects the intra-venous pressure in Schlemm's Canal.

   (*b*) Obstructive, when a diminished access of aqueous to the venous circulation tends to convert the intra-ocular circulation into a rigid one.

3. The principles of the mechanism of the vascular class are exactly the same as those that induce increased intra-cranial pressure.

4. The principles of the mechanism in the obstructive class are similar to those obtaining in the Primary disease, only differing in sclerosis of the Cribriform Ligament not being the agent.

Cupping of the Disc.

The function of the Lamina Cribrosa is that of a supporting framework to the fibres of the optic nerve as they pass into the retina, and so long as the intra-ocular and intra-cranial pressures stand at the same intra-venous level, it is never called upon to withstand any pressure. This unity of pressure holds good under all physiological conditions, but in pathological states the pressure in either eye or brain may rise independently. This pressure discontinuity in Glaucoma produces the well-known clinical feature of Cupping of the Disc. The essential pathological characteristic of this condition is retrocession of the Lamina Cribrosa, in the clinical manifestation of which the associated atrophy of the nerve fibres plays an altogether secondary part. The Cupping of the Disc is, I would maintain, an ectasia, and not an excavation of the nerve-head from atrophy—" cavernous " or otherwise.

Dissociation between the intra-ocular and intra-cranial pressures causes the Lamina Cribrosa to yield, and the resulting ectasia follows not so much from the height as from the long-continued disproportion of pressure, however slight, between the two sides of the nerve-head. As the converse to the position which the Lamina Cribrosa occupies in Glaucoma, one finds it arched in the opposite direction when the intra-cranial pressure is raised above the intra-ocular, provided this disparity of pressure has likewise been long enough established.

The various other pathological findings at the Disc in

Glaucoma are but an index of the clinical conditions at work producing the particular type of the disease from which the eye suffered, and therefore, like the exciting causal factors, these changes differ *inter se* in degree, but not in kind. Retrocession of the Lamina Cribrosa is the primary pathological feature in every case of sufficiently long standing, though upon it may be superimposed secondary manifestations depending on the clinical type of Glaucoma. Thus, in chronic non-congestive Glaucoma atrophy of the intra-ocular structures is a marked and prominent feature, and therefore the appearance of the ectasia or cupping is accentuated by the associated nerve atrophy. In chronic congestive Glaucoma, exudates analogous to the similar formations on the Iris surface may be thrown out, and these exudates by organizing and contracting may pull the retina into the cavity of the Disc. An acute congestive attack may, on the other hand, produce on pathological examination an appearance of "neuritis," in consequence of congestive œdema causing the nerve fibres to swell up and occupy all the available space formed by the retrocession of the Lamina Cribrosa. This condition of œdema is, of course, of the same nature as that existing throughout the intra-ocular tissues as the result of the altered conditions of the circulation calling forth an acute reaction on the part of the tissues.

All the manifold histological changes at the Disc can thus be correlated with each other, as well as with the manifestations of the primary disease in other regions of the intra-ocular contents, without having recourse to hypothecating any pathological peculiarity *sui generis* of the Disc structures.

Schnabel* regarded cupping of the disc as a true excavation produced by a "cavernous" atrophy of the optic nerve-head, and retrocession of the Lamina Cribrosa as a secondary pathological manifestation following atrophy of the Optic Nerve. This view has received the support, among others, of Schmidt-Rimpler.* There is, however, no necessity for hypothecating any such sequence of events; for the application of the ordinary hydrostatic laws meets every requirement, and explains in a rational and simple manner the existence of ectasia of the Lamina Cribrosa, as well as the presence of the associated atrophy. Both ectasia and atrophy are the result of the same common causes: a rise in the intra-ocular pressure above the normal intra-venous level.

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* Schnabel, *Archiv für Ophth.*, vol. xxiv., 1892.

The chief histological feature of the "cavernous" atrophy consists in the vessels lying above the niveau of the atrophic nerve fibres, so that one may obtain sections in which the vessels appear actually to traverse an open space or cavity in the optic nerve-head. This superficial position of the vessels is, however, just what one would expect from what has already been stated as to the vascular nature of the intra-ocular tension. The vessels stand above the level of the atrophic fibres for the simple reason that the hydrostatic pressure within them is never less than that without them. Whatever the intra-ocular pressure is, the intra-venous pressure must be the same, and thus the vessels only passively follow the retreating nerve elements as these spread out to line the bottom and sides of the cupping, formed by the funnel-shaped scleral opening of the Optic Nerve. As the retrocession of the Lamina Cribrosa increases, and atrophy of the nerve fibres becomes more marked, the vessels stand out more and more, not because of any peculiar "cavernous" atrophy, but only because the pressure in the arterial branches exceeds, and in the venous trunks is the same as, the intra-ocular pressure. The hydrostatic pressure within the eye firstly pushes back the Lamina Cribrosa, and secondly causes atrophy of the nerve elements; but the main vessels cannot be shoved out of the way, and so lie above the level of the atrophied fibres. Hence, the longer glaucoma has been established, the more superficial will the position of the vessels appear to be (Fig. 47).

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* "Graefe-Saemish," 2nd edit., Teil ii., Band vii.

The proper interpretation of the appearance of so-called "cavernous atrophy" can only be obtained, as in the case of the anterior segment of the eye, by the reconstruction of serial sections. By following out this procedure, the "atrophic cavities," seen in random sections of the Disc, are shown to owe their existence merely to the sectioning of the overhanging margins of the cupped Disc (Fig. 47).

A section running vertically through the nerve-head (*aa*), and striking the nerve fibres as they turn back to line the scleral foramen, will certainly give an appearance of "cavernous atrophy," but the true interpretation to be put to this appearance is perfectly apparent when serial sections are studied.

Fig. 47 represents a diagram reconstructed from serial sections (*bb*) of a cupped disc with vessels coursing on the surface of the atrophic nerve fibres. The upper half of the diagram represents the surface view.

The scleral opening for the optic nerve being funnel-shaped, as the Lamina Cribrosa has been pushed backwards, its circumference has been pressed against and clings to the sloping scleral walls, so that the Lamina appears as if hung from the edge of the foramen. The nerve fibres of the disc follow the retreating membrane, and so come to line the sloping sides of the cup; while, as already discussed, the bloodvessels only passively follow the nerve elements, and thus come to run on the surface. Now, sections which happen to run parallel with the sides of the scleral foramen will give rise to appearances which, unless serial sections are studied, may well give rise to the impression that we are dealing with a condition of "cavernous atrophy."

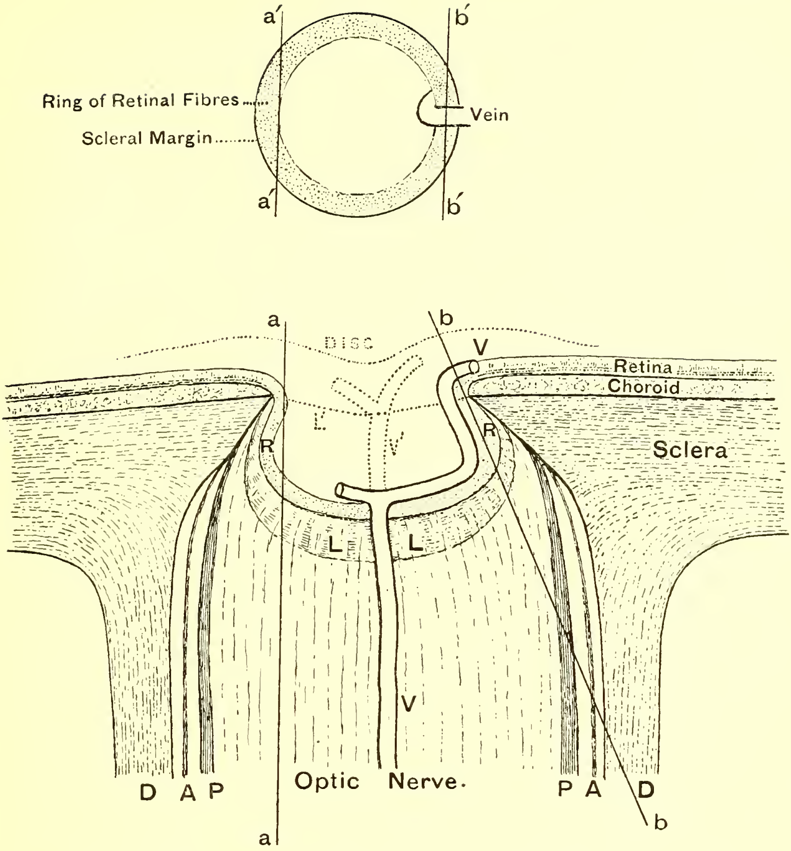

Fig. 47. — Reconstructed Diagram of Cupped Disc, from Serial Sections running in the Direction of the Line *bb*, Individual Sections of which show so-called "Cavernous Atrophy" of the Nerve-Head (see Fig. 49).

This appearance is purely artificial, produced by the way the sections are made.

The structures of the normal Disc are represented in dotted outline. The Lamina Cribrosa (L) in Glaucoma is driven backwards, and appears suspended from the margin of the Scleral Foramen, while the Retinal Structures (R) are pressed against the wall of the scleral opening. The Vessels (V) passively follow the retreating structures.

Thus, a vertical section (*aa*) through the edge of the Disc will present a large central cavity of apparent cavernous atrophy, which is, however, merely a part of the general space caused by the primary yielding of the Lamina Cribrosa.

The line *bb* represents the course of the section seen in Fig. 48.

The upper half of the diagram shows the Disc on surface view.

Fig. 48 represents Section 2 of the series from which Fig. 47 was reconstructed, and it shows apparent retraction of the Lamina Cribrosa without excavation. This section is not only a very peripheral one, but it runs obliquely parallel to the walls of the scleral foramen, and therefore merely goes through the nerve fibres as they line the cupped Disc. Fig. 49 represents Section 5, and the "cavity"

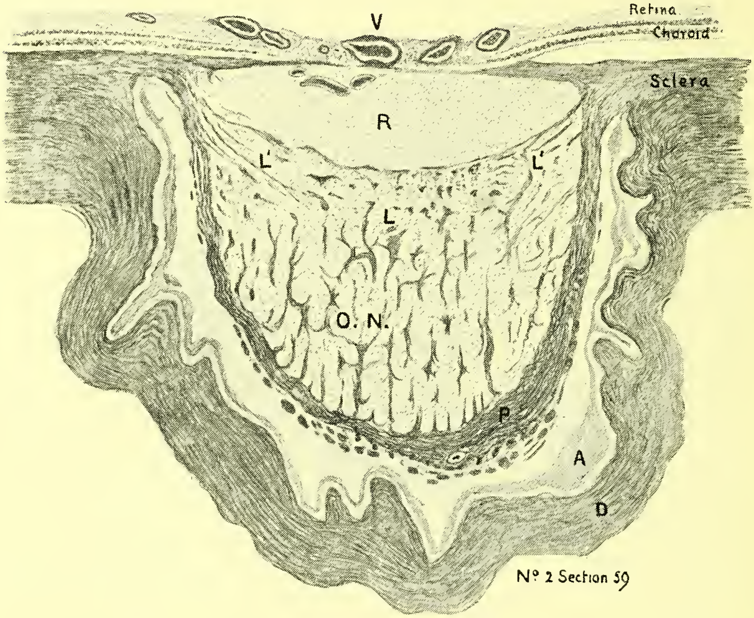

Fig. 48.—Section of Disc of Chronic Glaucoma, showing Retrocession of Lamina Cribrosa (L), with Apparent Absence of Cupping.

This is Section 2 of the series from which Fig. 47 was reconstructed, and marked *bb*.

The vein (V) is sectioned transversely. The retinal structures (R) are merely the nerve elements, which lie pressed up against the walls of the scleral foramen.

The fibres of the Lamina Cribrosa are sectioned transversely in the centre at L, and longitudinally at L′.

The fibres of the Optic Nerve (O.N.) are surrounded by their Pial (P), Arachnoid (A), and Dural (D) Sheaths.

present is simply part of the space of the cupped Disc, and is not due to any peculiar form of atrophy which the nerve-head has undergone, as is shown by the further sections

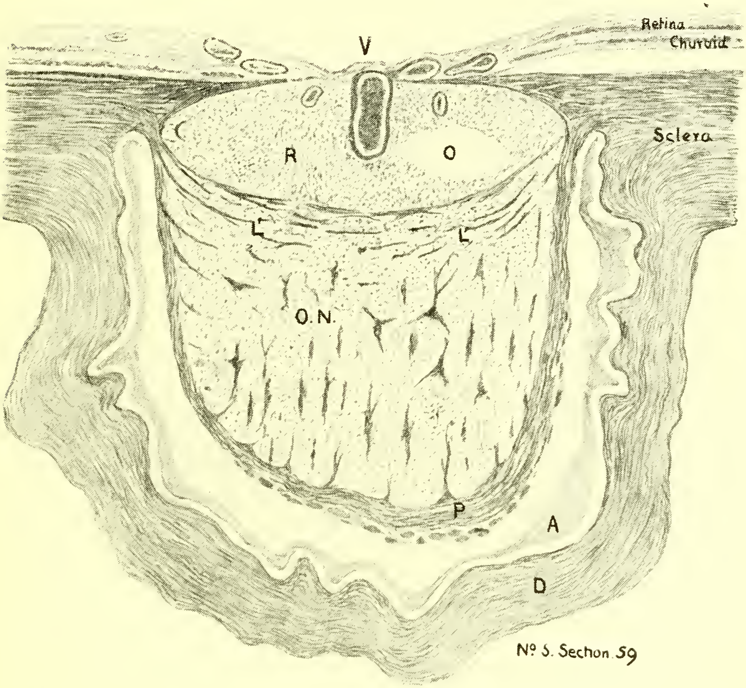

FIG. 49.—SECTION SHOWING SO-CALLED "CAVERNOUS ATROPHY" OF THE NERVE-HEAD.

This represents Section 5 of the series, which runs to the inner or left of the line *bb* of Fig. 47, and therefore the vein (V) is seen in longitudinal section.

The cavity (O) is formed by the sections beginning to miss the nerve elements lining the scleral foramen, and therefore now crossing a part of the general retrocession, of which this space is a part.

which go to make up the reconstructed diagram (Fig. 47). Fig. 48 is in every way comparable to the illustration given by Schmidt-Rimpler* as an example of "cavity formation without excavation of the papilla, but with retraction of the Lamina Cribrosa." Schmidt-Rimpler's section is obviously also an oblique one, for, as in my own cases, the optic nerve is very short, while its proximal end is surrounded by the vaginal sheath. Individual and random sections from my specimens of cupped disc of Glaucoma would apparently support the view of a "cavernous atrophy," but when studied in series they not only prove that no such condition exists, but they also demonstrate how the appearance arises.

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* Schmidt-Rimpler (see "Graefe-Saemish Handbook," p. 1000, Fig. 10).

PART IV

TREATMENT

Rationale of Treatment.

To appreciate the *modus operandi* of treatment in Primary Glaucoma, it is essential to bear in mind the vascular nature of the intra-ocular pressure, and the means by which the normal elastic circulatory system is converted into a rigid one by the obstruction created by sclerosis of the Cribriform Ligament. Rational treatment demands, therefore, that the passage of Aqueous to veins be facilitated.

This is exactly what iridectomy does, for by means of the raw surfaces created by the removal of a piece of Iris the Aqueous finds a ready passage to the Iris Veins whereby the incompressible intra-ocular fluids cease to act as a rigid volume. A Glaucoma Iridectomy acts in the same way as an incision in the rubber tubing at I in the "Glaucoma" flask (Fig. 39). Such an opening has no influence on the volume of the Flask contents, which must always be constant whatever the internal pressure may be; but the opening prevents the incompressible fluids acting as a rigid mass, and so converting the elastic walls of the tubing into a rigid system. With free communication restored, the mechanical conditions in the "Glaucoma" flask become, in fact, the same as those which obtain in the flask in which the openings C are uncovered (Fig. 37).

Exactly the same relationship exists between the normal eye and that with an effective Glaucoma Iridectomy, as in both there is free and sufficient contact between intra-ocular fluids and veins.

In Primary Glaucoma the passage of Aqueous to Schlemm's Canal is obstructed, and so the main or only access to the intra-ocular veins is through the Iris Crypts. If such passage is not sufficient, the intra-ocular circulation tends to be converted into a rigid system, and the pressure rises. Iridectomy effects the formation, so to speak, of a large open crypt presenting three sides of a square, the raw surface of which allows of the free passage of Aqueous to the Iris Veins. The primary obstruction presented by sclerosis of Cribriform Ligament is thus counterbalanced, and as the circulation resumes its elastic nature the intra-ocular pressure falls again to the normal intra-venous level.

The principles involved in the action of Myotics are fundamentally the same as in Iridectomy, for contraction of the pupil, by opening out the Iris Crypts, likewise facilitates the passage of aqueous to the Iris Veins. This action is, however, merely temporary, lasting only for so long as the pupil remains contracted; while, on the other hand, the effect of an Iridectomy is permanent, as the raw edges of a post-operative coloboma always remain open, provided the Iris Stroma is not sclerosed and atrophied. Myotics, besides their more limited benefits, tend to defeat their object by causing congestion and irritation of the eye, and so increasing the vascular and intra-ocular pressures.

The vascular nature of the intra-ocular pressure explains why Iridectomy and Myotics, though having such markedly beneficial effects in Primary Glaucoma, have no influence on the normal intra-ocular pressure.

The intra-ocular pressure represents the intra-venous pressure in the eye, and therefore widening of the existing area of contact between the Aqueous and intra-ocular veins, either by Iridectomy or by maintaining the Iris Crypts open with Myotics, cannot be expected to affect the intra-ocular pressure in the slightest.

The intra-ocular pressure must always remain at the level of the intra-venous pressure, as being the lowest pressure existing in the eye, and below this pressure that of the intra-ocular fluid contents cannot fall.

However free the contact between the aqueous and venous walls may be, this hydrostatic balance must always be maintained, provided the eyeball remains a closed sphere. Exactly the same condition is to be met with in the model illustrating the circulatory nature of the normal intra-ocular pressure (Fig. 37), if the rubber tubing at I is cut so as to increase still further the contact between the circulatory fluid and the water in the flask. This extra opening would represent an Iridectomy in an eye with normal tension, and its effect on the pressure in the flask is *nil*, as it will still remain at 15 millimetres Hg, as representing the outflow or venous pressure at V.

A similiar incision in the rubber tubing of the "Glaucoma" flask (Fig. 39), by restoring the connection between the circulatory and the contained fluids, will correspond to the action of Iridectomy in Glaucoma. The pressure in the flask will be unable to rise above 15 millimetres Hg, nor will it be able to fall below this level, provided the pumping force is the same.

Thus, not only do these working models admirably illustrate the hydrostatic principles involved in the circulatory nature of the intra-ocular pressure, but their value is further enhanced by the simple demonstration they give of the *modus operandi* of Iridectomy.

General Medical Treatment.

The remedial measures of Iridectomy and Myotics only influence the constant mechanical and predisposing factor of Primary Glaucoma, and leave altogether untouched the exciting vascular element of the disease. It is in this latter direction that I venture to consider more thought and recognition will be given in the future than has been the case in the past.

The importance of general medical treatment lies in maintaining a low level of general circulatory pressure, or at any rate in preventing a high one. The intra-ocular pressure is influenced, as previously discussed, by changes in the general arterial or general venous pressures, whereby a rise in general arterial pressure induces a proportionate rise in the intra-ocular pressure; while the effect of an increased general venous pressure is a much more direct one, producing millimetre for millimetre a corresponding increase in the intra-ocular pressure.

These physiological considerations, while novel, receive ample confirmation in the present purely empirical management of an acute glaucomatous attack, although the application of these same principles can well be extended beyond the range of mere active intervention to embrace measures of preventive treatment.

Summary of Rationale of Treatment.

1. Primary Glaucoma follows from diminished access of Aqueous to Veins, resulting in the intra-ocular circulation being converted into a rigid system.
2. Treatment to be effective must facilitate the passage of Aqueous to Veins, and so restore the circulation to its normal elastic nature.
3. The beneficial results of Iridectomy follow in consequence of the raw edges of the coloboma permitting access of the Aqueous to the Iris Veins.
4. Myotics, by contracting the Pupil and opening the Iris Crypts, act in the same manner.
5. Iridectomy and Myotics cannot influence the normal intra-ocular pressure, as such already represents the lowest circulatory pressure in the eye, and further contact

between Aqueous and Veins cannot reduce it below this level.

6. Mechanically an Iridectomy acts in the same manner as an incision at I in the rubber tubing of the "Glaucoma" flask (Fig. 39).

Iris Wounds.

The most striking histological feature exhibited by the healthy Iris after Iridectomy is the extraordinary amount of trauma that its tissue presents without any manifestations whatsoever of subsequent reaction or cicatrization. Histologically the normal and healthy iris tissue is absolutely indifferent to trauma as such, so long as it is not subjected to toxic agencies (Figs. 50 to 55). Under normal circumstances the Iris tissue undergoes no reparative processes, so that no scar is formed when its tissue is divided or torn, and no connective tissue is laid down when it is pulled from its attachment to the Ciliary Body; and, similarly, no reaction follows traumatic hæmorrhages into its substance. The so-called "anterior endothelium" of the Iris has no tendency to cover the wound, or the pigment layer to grow over it, so that, with the single exception of the absorption of extravasations, the iris tissue appears years after an Iridectomy exactly as it did immediately after its performance—in fact, just as if the operation had been performed post-mortem. Thus, the severed Iris tissue forming the base and pillars of the coloboma always remains an open, raw, and unhealed area, in free and open communication with the aqueous of the anterior chamber, in exactly the same manner as the crypt surfaces.

In performing Iridectomy it is not only that part of the Iris pulled through the Incision which is subjected to traction, but the whole Iris structure is stretched, and feels the resulting strain. As the pupillary margin is drawn upwards by the forceps, the strain is transmitted to the whole circumference of the sphincter pupillæ as well as to the ciliary attachment of the iris periphery, the consequence of which is that ruptures of its surfaces, both anterior and posterior, at the root of the iris stump forming the coloboma, as well as in the rest of the iris circumference, are commonly met with in microscopic sections. These rents and lacerations can be looked upon as a series of artificial and accessory crypts which assist the raw area of the base

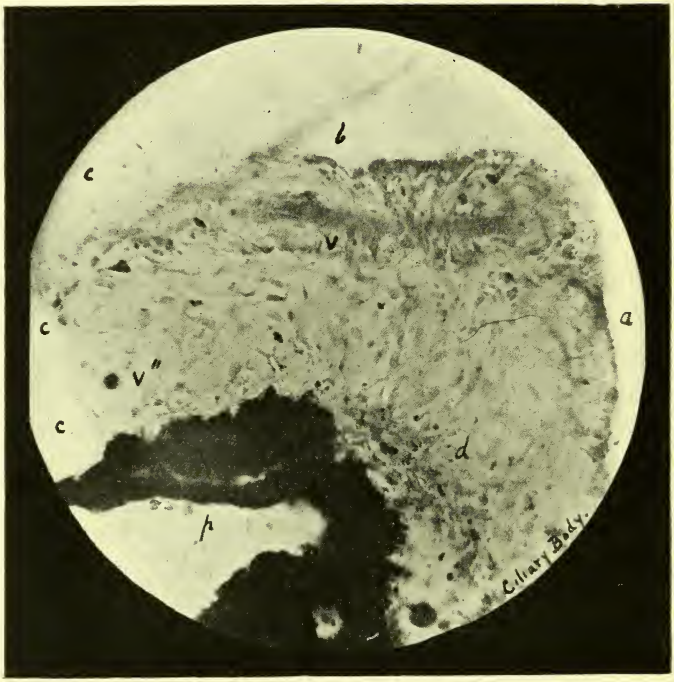

Fig. 50 —Iris Stump Seven Days after Extraction. From a Case of Professor Fuchs's.

The length of Iris Stump is unusually small, only 0·35 millimetre.

Angle of Anterior Chamber (*a*); Anterior Iris Surface (*b*); Cut Surface of Iris (*c*, *c*, *c*), forming the base of the Coloboma; Posterior Pigment Layers (*p*); Vessel in longitudinal (*v*), and in transverse (*v″*), section; Origin of Dilator Pupillæ fibres (*d*).

and pillars of the coloboma in bringing the aqueous to the iris veins.

That the normal Iris after Iridectomy should not, even years after, show any signs of scar or other manifestations of reaction might appear at first sight incredible, from what we know of the changes which follow wounds in other structures of the body, although, when we appreciate the unique conditions under which the tissue cells of the Iris lie, it will be found that this lack of cicatrization has a perfectly physiological basis.

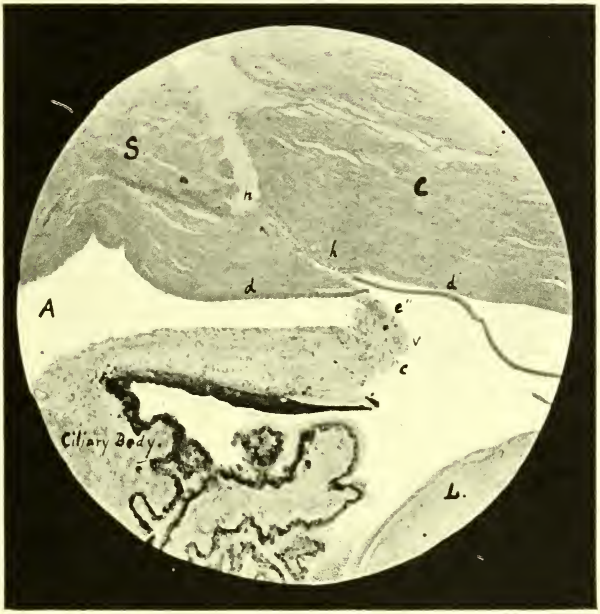

Fig. 51.—Iris Stump Fifteen Days after Extraction. From a Case of Professor Fuchs's.

The cut surface of iris (*c*) is cut obliquely, so that the Anterior aspect is longer than the Posterior. At *c*″ there is an extravasation of blood. Vessel (*v*) cut transversely projecting into Anterior Chamber. Scleral (S) and Corneal (C) surfaces of incision (*h*) ; A, Angle of the Anterior Chamber ; L, Lens Capsule enclosing cataractous débris.

The reaction of a tissue is the response of its cellular elements to stimuli of whatever nature, and cicatrization is the final expression of this process.

When a break is made in the continuity of a tissue, as by an incision in the skin, the conditions of cellular life of the cells bounding the wound are profoundly altered. It is this alteration of cellular life in the part involved that finds

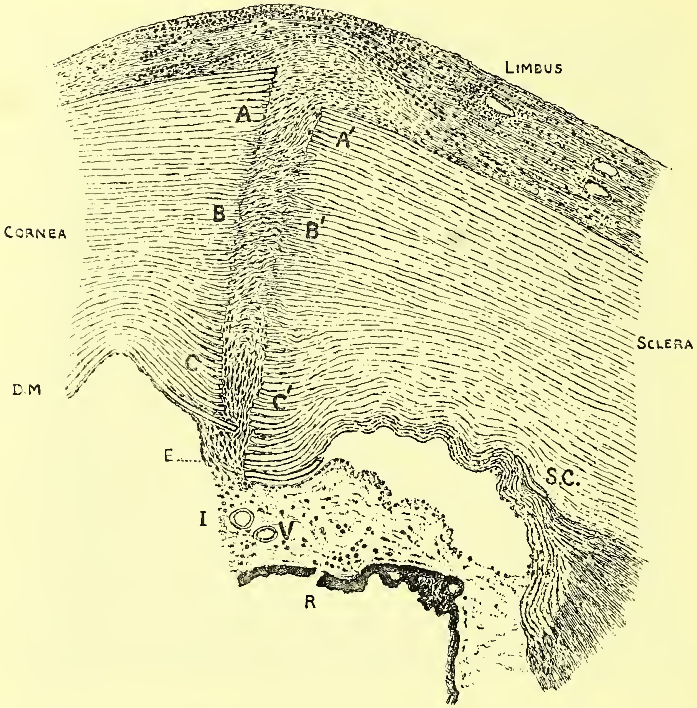

FIG. 52.—IRIDECTOMY STUMP AFTER EXTRACTION. POST-MORTEM CASE ; DATE UNKNOWN.

The Incision (A,B,C) is not completely cicatrized. There is in this section an adhesion of the Iris (I), the cut surface of which manifests no reparative changes. Iris veins (V) in cross-section ; there is a rent (R) in the Posterior Pigment Layers. Descemet's Membrane (D.M.), with its endothelium (E), which has relined the inner extremity of the incision, but stops short at the raw Iris Surface.

expression in cicatrization. Now in the case of the human Iris the conditions of its cellular life are unique, for its tissue is not a dense structure, but a loose stroma immersed in a lymph fluid, which fluid, owing to Fuchs's crypts, bathes and surrounds all the tissue elements. The Iris is, in fact, so situated that any break in the continuity of its structure, as by Iridectomy, does not alter in the slightest the con-

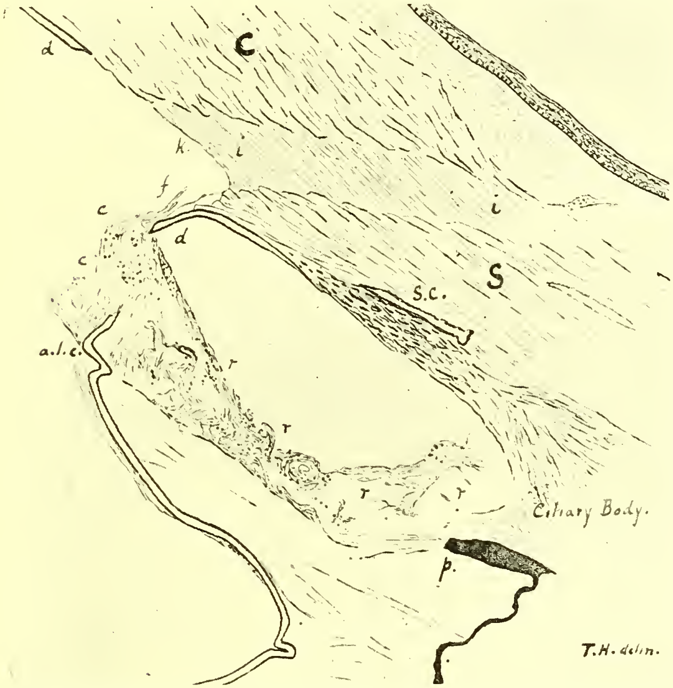

Fig. 53.—Iridectomy Stump after Graefe's Extraction. Case of Professor Fuchs; Date unknown.

The incision (*i*) has healed, with the interposition of a large intercalary mass between the Corneal (C) and the Scleral (S) margins of wound.

The Iris Stump is denuded altogether of the Posterior Pigment Layers (*p*), and shows numerous rents and lacerations (*r*).

The cut surface (*c*) shows no cicatrization; at *f* there is an attachment of Iris to Descemet's Membrane (*d*).

The remnant of the Anterior Lens Capsule (*a.l.c.*) is adherent to the Iris Stump.

dition of cellular life of the cells bounding and forming the wound.

An Iridectomy simply removes and separates adjoining cells, but the condition of cellular life of the cells left behind is in no way altered, as they still remain bathed by aqueous, and therefore no stimulus is imparted to the tissue cells to bring about cicatrization. It is thus that,

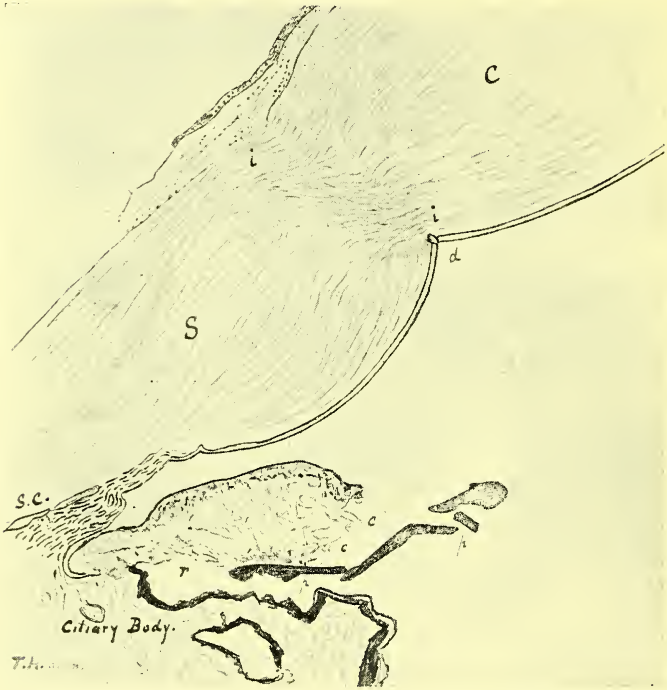

Fig. 54.—Iridectomy Stump Four Years after Graefe's Extraction. Case of Professor Fuchs.

The line of the incision runs through *i*, *i*; at *d* the several ends of Descemet's Membrane are united by a new formation of homogeneous membrane.

The Iris Stump has its cut surface (*c*) severed obliquely. Entire absence of any signs of cicatrization.

On the posterior aspect there is a rent (*r*) of the Posterior Pigment Layers, which hang down as a long tag (*pp*) into the Anterior Chamber.

under modern and aseptic conditions, the Iris is found in the same state years after an Iridectomy as if such had been performed post mortem, provided the Iris at the same time of operating was normal and healthy. This last is a most important practical point.

The posterior pigment layers of the Iris under normal circumstances play the same passive part after Iridectomy as the other structures entering into the formation of the Iris, for, as already mentioned, they have no tendency to grow over the wound surface. The *pars iridica retinæ* is, in fact, never regenerated, and remains in the same state years after Iridectomy as immediately subsequent to the operation, for it is never replaced, even when rubbed off by the lens in the act of extraction. Similarly, when the Iris stump is pulled out from its attachment, leaving the anterior part of the Ciliary Body bare, this denuded surface shows no more signs of reaction than the Iris tissue itself; for no connective tissue is laid down, nor does the pigmented epithelium of the *pars ciliaris retinæ* tend to grow over the rent surface.

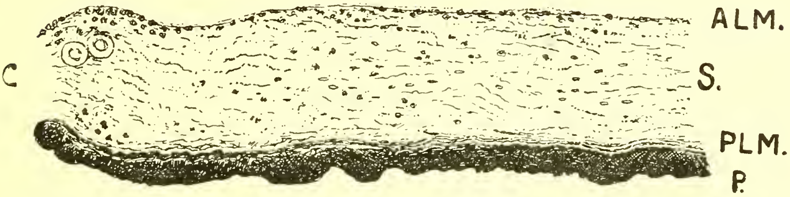

FIG. 55.—PILLAR OF COLOBOMA TWO YEARS AFTER IRIDECTOMY. CASE OF PROFESSOR FUCHS.

The iridectomy was done four months after a simple extraction, in consequence of a blow in the eye after leaving hospital. The patient died two years later.

The section shows absence of any healing or reaction in the cut surface (C.) of the loose Iris stroma (S.). The Anterior Limiting Membrane (A.L.M.) terminates abruptly at the edge of the coloboma, as likewise do the Posterior Limiting Membrane (P.L.M.) and the Pigmented Epithelial Layers (P.).

Iridectomy in Chronic Glaucoma.

In Chronic Glaucoma the Iris tissue has frequently undergone more or less extensive alteration before the condition comes under the surgeon's care, and therefore the results obtained by Iridectomy are apt to be as uncertain as they are brilliant in Acute Glaucoma. The only assured clinical condition we have to guide us as to the probable effect of operation is the state of the Iris tissue, which, if it has already undergone atrophy, is sure in the end to render treatment unavailing. Atrophy and sclerosis are the product of the reaction of the Iris tissue to the altered conditions of cellular life consequent on the long-continued, though not necessarily high, hypertonia. The loose Iris stroma becomes thereby altered, and replaced by a more or less dense fibrous structure. The excision of a piece of such an Iris will, thus, not materially facilitate the passage of Aqueous to the Iris Veins; for not only does the sclerosed state of the Iris tissue at the outset hinder the passage of Aqueous to the Iris Veins, but also, as the original process of fibrosis, once started, cannot be undone, the cut surfaces of the Coloboma tend in time to undergo contraction, so that what should be a raw and open area eventually becomes sealed up (Figs. 56 and 57). The fibrosis of the Iris Stroma in Chronic Glaucoma leads to occlusion of the natural Iris crypts, and the similar changes at work tend ultimately to closure of the operative crypts formed by Iridectomy. In this way the immediate beneficial results are counteracted in the course of some months or years.

The inefficacy of Iridectomy in Chronic Glaucoma is ascribed to the difficulty of removing the firmly adherent Iris Root, but that such cannot be the true cause of failure is shown by the consideration that sclerosis of the Cribriform Ligament precedes the angular occlusion by the Iris Root, so that Glaucoma exists, altogether apart from this pathological condition. I have further had the opportunity of examining three eyes in which Iridectomy had been faultlessly performed by Richardson Cross, and the Iris

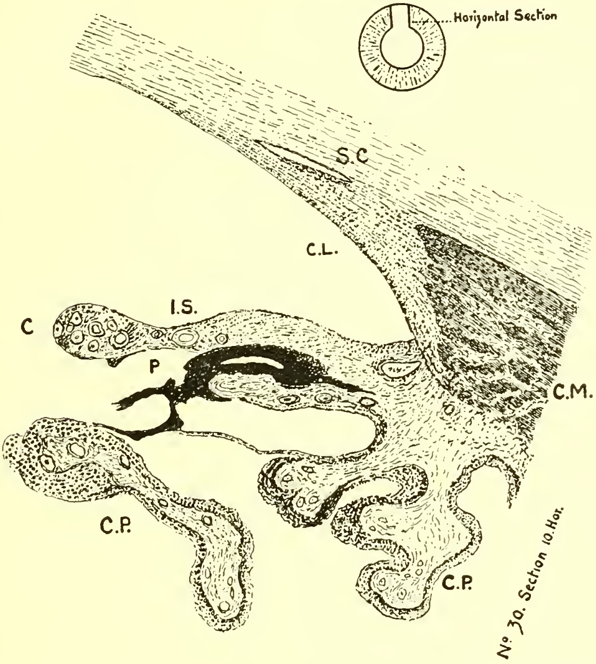

Fig. 56.—Horizontal Section of Pillar of Coloboma in Chronic Glaucoma.

The inset shows position of section. History of case lost. Disc deeply cupped. The Angle of Anterior Chamber throughout circumference not occluded.

Schlemm's Canal (S.C.), on the inner side of which lies the sclerosed Cribriform Ligament (C.L.) of the Ciliary Muscle (C.M.).

The Iris Stroma (I.S.) is compact and fibrous, and at the margin of the Coloboma (C.) it shows a knoblike thickening, which contains a number of large new formed vessels surrounded by fibrous tissue. The cut area of the Iris tissue is thus sealed up. A portion of the posterior Iris surface is denuded of its pigment epithelium (P.).

The Ciliary Processes (C.P.) are sclerosed and atrophic. The one to the right is sectioned more or less longitudinally, while that seen to the left is cut transversely. The outer layer of epithelial cells of the processes shows the complete absence of pigmentation characteristic of a senile eye.

Root removed up to its extreme limit, leaving the upper half, and in one case the whole circumference, of the Angle of the Anterior Chamber free, and yet in all these cases the ultimate and practical results of operation were futile (Figs. 56 and 57).

Summary of Changes following Iris Wounds.

1. Wounds of the healthy Iris never manifest any cicatricial changes.

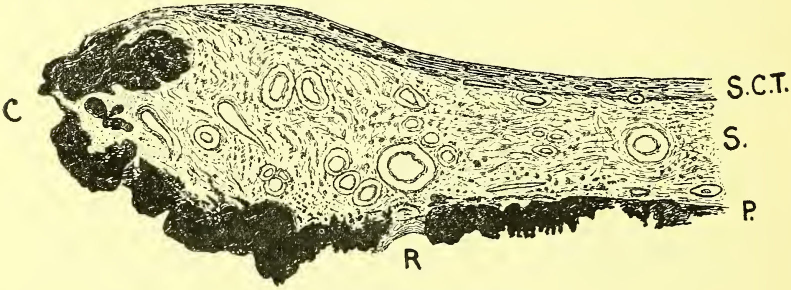

Fig. 57.—Horizontal Section of Coloboma in Chronic Glaucoma. Case of Mr. Richardson Cross.

The position of section is the same as in Fig. 56. Iridectomy performed eight years before the eye became blind and painful, and had to be removed. The Angle of the Anterior Chamber open.

The Iris Stroma (S.) is markedly sclerosed, and on its surface there is a thick superficial connective-tissue formation (S.C.T.), with many new-formed vessels.

As in Fig. 56, a cicatricial formation of the Iris tissue entirely closes the Iridectomy wound, and gives a knoblike appearance to the margin of the Coloboma (C.).

The Pigment Epithelium (P.) shows a rent (R.) which is closed by connective tissue.

2. Traction on the Iris in performing Iridectomy causes minute rents and lacerations throughout its circumference. These "traumatic crypts" assist the raw edges of the Coloboma in bringing the Aqueous to the Iris veins.

3. The absence of reaction to trauma on the part of the healthy Iris tissue is due to its loose stroma being con-

stantly flooded by Aqueous, so that the condition of its cellular life remains unaffected.

4. The atrophy and sclerosis of the Iris in Chronic Glaucoma are an expression of reaction of its tissue to the altered conditions of the circulation.

5. In Chronic Glaucoma, if the Iris stroma has already undergone sclerosis, this will not only hinder the direct contact which should take place between Aqueous and Veins, but will lead ultimately to contraction and sealing up of the Iris Wounds.

Glaucoma following Extraction with Iridectomy.

The view that the open state of wounds in the healthy Iris is the means by which the Aqueous in Glaucoma obtains a wider admission to veins, and so prevents the continuance of the raised pressure, naturally at once raises the question of Glaucoma following extraction with Iridectomy; for it might be objected, with apparent reason, that if this explanation were correct, then certainly increase of tension should not follow in such cases.

Now, the senile and physiological sclerosis of the Cribriform Ligament of the Aphakic eye differs merely in degree from that met with in Glaucoma, and therefore Iridectomy is admittedly a positive help by facilitating the access of Aqueous to the Iris Veins. This is, after all, only what Iridectomy can claim to do, if the Iris is healthy and remains so; but Iridectomy cannot control a rise of intra-ocular pressure induced primarily by vascular changes. If the intra-ocular circulatory pressure is raised by an inflammatory state of the Iris or Ciliary Body, then the intra-ocular pressure is raised quite irrespective of how free the contact between aqueous and veins may be, for intra-venous pressure and intra-ocular pressure stand and move together. It is in such a manner that Glaucoma may arise secondary to Iritis during the first few weeks or months after operation. But, on the other hand, while congestion or iritis may not be sufficiently acute to lead to Glaucoma, they may at any rate initiate changes in the Iris stroma which will lead in time to closure of the original raw surface of the Coloboma, and so make the Iridectomy functionally useless. Thus, bearing in mind the senile sclerosis of the Cribriform Ligament, and the changes which the surface of Iris wounds may undergo in congestive states of the Iris, we can realize how, in spite of an Iridectomy, Glaucoma, with all the clinical and pathological manifestations of the primary disease, may arise after extraction, either soon after the operation or after a delay of several years.

Much has been made of the pathological findings of Iris prolapse, or adhesion of capsule to the cicatrix, as tending to initiate occlusion of the entire circumference of the Angle, and so produce Glaucoma ; though here again, from what has been already stated, it must appear that, as in other forms of Glaucoma, the occlusion is a secondary pathological manifestation, the result of "congestive œdema."

Iris prolapse or capsular entanglement in the wound may play a part in inducing Glaucoma, but certainly not in the manner assumed in current literature. Cicatricial adhesions cannot by themselves start Glaucoma, but they may, by causing traction on the Ciliary Body, undoubtedly induce congestive changes within the eye, which will start a sequence of events, such as already enumerated, in which occlusion of the Angle is the result of "congestive œdema," but not the cause of the Glaucomatous onset.

Post-Operative Ciliary Detachment.

This form of Detachment was first described both clinically and pathologically by Fuchs.* In most cases the condition appears two to eight days after operation, and may last as long as a month, or even longer. The clinical sign which arouses suspicion is a shallow Anterior Chamber. Fuchs ascribes the occurrence of this post-operative detachment of the Ciliary Body and Choroid to the rents and laceration of the circumference of the Iris Root, which, as already described, form one of the chief histological features characterizing the Iris after Iridectomy. These microscopic ruptures, he holds, produce free and open communications between the Aqueous in the Anterior Chamber and the tissues of the Ciliary Body, whereby fluid is aspirated into the Supra-Choroidal Space.

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* Fuchs, *Archiv für Opth.*, vol. liii.

The difficulty of accepting this explanation of aspiration lies in the fact, already demonstrated, that the fluid normally present in the Supra-Choroidal Space is derived from the Aqueous percolating through the fasciculi of origin of the Ciliary Muscle, in consequence of which the same hydrostatic pressure exists in the Supra-Choroidal Space as elsewhere throughout the intra-ocular fluid contents. To this must be added the further consideration that the presence of crypts on its surface precludes the Iris being regarded in any way as a water-tight structure, such as the hypothesis of aspiration would demand; while, finally, I would urge against this view that the cut surfaces forming the base and pillars of the Coloboma always remain open and uncicatrized areas which present a very much larger absorbent surface than the small microscopic rents created by the unavoidable traction in performing Iridectomy. Individually and collectively these various considerations militate against the aspiration theory of the Detachment.

The principal and characteristic clinical feature of post-operative serous detachment of the Choroid is a shallow Anterior Chamber, the result of delay in coaptation or union of the lips of the wound. So long as the aqueous escapes through the incision, the aqueous and intra-venous pressures do not balance each other, and therefore the fluid formed under these conditions is a true filtrate containing a considerable proteid percentage.

Like the intra-cranial volume, the intra-ocular volume is fixed, so that loss of Aqueous volume is replaced by increase in the vascular volume, particularly in the Uveal tract.

The loss of volume in front of the Lens becomes thus replaced by a corresponding vascular volume behind it, so that the lens, or in the case of extraction the posterior lens capsule, advances, while the disturbed vascular relationship results in the pouring out from the vessels of the Iris and Ciliary Regions of a serous transudate, which will collect in the Anterior part of the Supra-Choroidal Space as a passive collection of fluid. As soon as the corneal wound becomes sufficiently sealed so as to withstand the intra-venous level of the normal pressure of the intra-ocular fluid contents, the Anterior Chamber is re-formed, and the fluid in the Supra-Choroidal Space becomes resorbed. The speedy disappearance of this serous fluid on the restoration of the Anterior Chamber is another factor pointing to the circulatory and physiological nature of this form of detachment, and disproving its formation by aspiration.

Theories of Iridectomy.

The number and diversity of the views which have been put forward to account for the mode of action of Glaucoma Iridectomy bear witness to the absence of a comprehensive explanation of the disease.

Retention theories of Glaucoma at present hold the field, and therefore it is but natural that operative measures should be held to succeed by relieving the retained intra-ocular volume. To establish such hypothesis it is necessary to be assured of its fundamental physiological basis.

Now, this whole work has been directed to showing that the intra-ocular pressure is not a question of balance of volume between inflow and outflow of aqueous, but that it stands with the intra-cranial pressure at intra-venous level; and from this fact it has further been shown that whatever the intra-ocular pressure may be, whether physiological or pathological, the intra-venous pressure must follow the same level. Owing to this absence of any difference in hydrostatic level between the aqueous and the intra-venous pressure, the aqueous outflow must be a diffusion or osmosis, and not a filtration.

From the pathological aspect, therefore, the issue is whether the pressure in Glaucoma must be regarded as the result of retained intra-ocular volume or of raised circulatory pressure, the volume being constant.

It would be futile to attempt to examine the different views held respecting the mode of action of Iridectomy, or of the various other means adopted to relieve Glaucoma, without appreciating the fundamentally different standpoints between the theory of retained intra-ocular volume on the one hand, and the vascular conception on the other; for the *modus operandi* of treatment invoked for the former theory is absolutely opposed to and untenable for the latter.

If Primary Glaucoma is not a question of retained volume, all theories of treatment based on increasing filtration are rendered untenable; while if Glaucoma follows from diminished access of aqueous to veins, whereby the elastic circulatory system in the unyielding globe is converted into a rigid system, then rational treatment demands that contact with veins be facilitated, by which means alone can the circulatory channels be restored to their elastic nature.

Treacher Collins* states "that the Iris Root tears away from its extreme root, thus leaving a large portion of the filtration area free from drainage, even should the remainder of the Iris retain its faulty position."

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* Treacher Collins, "Roy. Lond. Oph. Hosp. Reports," vol. xiii., 1891.

This view of the operation is one which is apparently supported by a certain amount of pathological evidence, for, in cases where Iridectomy has failed to give relief, subsequent examination has revealed the Iris Root, forming the base of the Coloboma, adherent to the back of the Cornea.

Removal of a portion of the adherent Iris Root does not, however, diminish the primary obstruction created by the sclerosed condition of the Cribriform Ligament; for, as already referred to in discussing the bearing of Iridectomy on Chronic Glaucoma, I have sectioned three eyes in which the requirements of a successful Iridectomy, as laid down by Treacher Collins, were fully complied with, and yet in all three cases the eyes became blind and painful, and had to be removed (Figs. 56 and 57).

It is cases like these that have given ground for the belief that it is not the Iridectomy, as such, which reduces the intra-ocular pressure, but that success depends on the operative incision. In pursuance of this theory, operations have been devised in which, if a piece of Iris is excised, it is only with the idea of preventing prolapse.

As is, unfortunately, too often the case when a theory has to be upheld, statements are apt to be made, in support of it, which are in complete disregard of correlated conditions. As an example of such contradictions, one finds, on this question of the incision for Iridectomy, that Parsons, although describing Schlemm's Canal as a plexus of veins,* yet states that the object of the peripheral incision in Iridectomy for Glaucoma,† as well as the aim of Sclerotomy,‡ is to open the filtration Angle and Canal of Schlemm.

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* Parsons, "Pathology of the Eye," p. 959.

† *Op. cit.*, p. 1110.

‡ *Op. cit.*, p. 1111.

Such procedure, if successful, would merely result in phlebotomy, with certainly most undesirable consequences.

Another theory of the *modus operandi* of Iridectomy incisions is that the cicatrix acts as a filter which allows of the escape of the pent-up aqueous. This view, first put forward by De Wecker,* has been revived by Lagrange,† and has led during recent years to the introduction of a great number and variety of operative procedures in the form of Sclerectomy, with or without Iridectomy.

Lagrange ascribes the benefit of Iridectomy in every form of Primary Glaucoma to the production of a permeable cicatrix, for he holds that "the filtering capacity of the cicatrix of an ordinary Iridectomy depends directly upon the degree of the hypertension of the eye that has been operated on, and therefore Iridectomy will be so much the more efficacious the more distended the eye."

We have thus before us the clear issues upon which must stand or fall the theory of filtering cicatrices, of which the first and foremost is the volumetric conception of the intra-ocular pressure.

If the intra-ocular pressure is not a question of fluid volume, but of fluid pressure, and Glaucoma not the result of retained volume, but of retained pressure similar to that in the "Glaucoma" flask (Fig. 39), then the whole *raison d'être* of filtration must fail.

Apart from this, however, a consideration of the principles of hydrostatics makes the above-quoted statements perfectly untenable; for if cicatrization of Iridectomy wounds were ordinarily to take place under pressure, and if this post-operative pressure were the more marked the acuter the Glaucoma, then it is a physical impossibility to hold that the intra-ocular pressure can be reduced, for the hydrostatic force cannot be expected to limit its energies solely to the cicatrix without at the same time transmitting the same force throughout the whole of the intra-ocular contents.

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* De Wecker, "Ther. Ocul.," i. 381.

† Lagrange, *Archives d'Ophtalmologie*, February, 1908. See translation, *The Ophthalmoscope*, 1908, p. 363.

Lagrange, in his enthusiasm for the application of the mechanical principle of filtration to Iridectomy incisions, has quite overlooked the fundamental law of hydrostatics, that fluids transmit pressure equally in all directions. If it is hypertension that keeps the lips of the wound open, then this same pressure must be acting throughout the intra-ocular contents, and therefore the intra-ocular pressure must be the same as that raised pressure which prevents coaptation of the margins of the incision. The law of hydrostatics, in short, reduces the theory of distension and filtration of Iridectomy wounds to a *reductio ad absurdum*, for how can one expect the pressure in an eye with glaucoma to be reduced to normal limits if it is the high pressure *after* the operation which forces the lips of the scleral wound apart?

The histological aspect of "filtering cicatrices" I have already dealt with in a paper which appeared in *The Ophthalmoscope*,* and therefore I do not propose to discuss the question again here; for, after all, interpreting such evidence as one will, it must naturally play a subsidiary part, for eventually one is always bound to be driven back to the consideration of the two incompatible views of the nature of the intra-ocular pressure.

If the aqueous can gain a lower hydrostatic level by filtration, it will do so, but then the physiological parity between aqueous and venous pressure will be destroyed.

We are thus led to a perfect impasse from which there is no escaping, for if the aqueous drains away through a "filtering cicatrix," how are the aqueous and venous pressures to be balanced? If not balanced, how are the elastic venous walls to be prevented from dilating, and producing stasis with absence of the anterior chamber such as always accompanies an obviously open wound?* How, in short, can the volumetric conception of the intra-ocular tension be squared with the view of a fixed intra-ocular volume whose pressure varies with intra-venous pressure?

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* "On so called Filtering Cicatrices in the Treatment of Glaucoma" (*The Ophthalmoscope*, December, 1907).

The theory of "filtering cicatrices" rests on the assumption that the aqueous outflow is a filtration, and that the intra-ocular pressure is the product of the volume of the contents of the globe; and therefore if these essential premises are correct, one may be forced to admit the feasibility of "filtering cicatrices," but not otherwise: for however logical deductions may be, if the essential premises on which they are based are at fault, the conclusions derived therefrom must be equally fallacious. In fact, the more logical the deductions, the more erroneous will be the final results. Now, the bases of the volumetric and of the circulatory conceptions of the intra-ocular pressure are as diametrically opposed as the two poles, and therefore, while admittedly the theory of "filtering cicatrices" is perfectly logical with the former premises, it is equally illogical with the latter.

Relation of the Intra-Ocular Pressure to Healing Wounds.

A wound in the stages of mediate and of primary union does not give way before the normal intra-ocular pressure, as the only strain which it is called upon to support corresponds to the level of the intra-venous pressure. If the intra-ocular pressure were the product of intra-ocular volume, it is inconceivable how a corneal wound could ever become properly united if it had constantly to bear the brunt of an increasing hydrostatic volume behind it; for the region of the incision, being the weakest spot in the eyeball, would invariably give way and bulge out before the ever-increasing volume of the intra-ocular contents. When we realize, however, that the cubic capacity of the eyeball is fixed, and that its contents lie at intra-venous pressure, we can quite understand how the fibrinous exudate that brings about primary union of a wound is sufficient to retain the aqueous volume. All the subsequent reparative changes take place at this normal intra-venous level of pressure, and it is only when the intra-ocular pressure rises above this that the wound will yield and protrude as a hernia or cystoid cicatrix.

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* See p. 146: Paracentesis.

A cystoid cicatrix thus implies that the intra-ocular pressure is standing above the normal intra-venous level, for otherwise the protrusion could not exist.

It has already been shown that what we recognize by palpation as the normal intra-ocular pressure is the intra-venous pressure of the fixed intra-ocular volume through the resistance imparted by the scleral tunic. With an open wound the eye feels soft, as the intra-ocular pressure is reduced to zero, and the resistance of the scleral tunic is destroyed by the incision. In the stages of mediate or of primary union the tension of the eyeball still feels subnormal, although the intra-ocular pressure corresponds to the intra-venous level, because the resistance of the fibrous tunic is not restored till the final stage of cicatrization is complete; for the sense of resistance which a closed sphere like that of the eyeball will impart on palpation depends on the resistance of its weakest spot. Thus it arises that an eye with a healing wound feels soft although its contents lie at the normal intra-venous level, while in the case of a cystoid cicatrix the tension may not be recognized as above normal although the contents pressure must be above normal intra-venous level.

The presence of a cystoid cicatrix cannot benefit an eye by bringing about filtration, as is commonly held, for I would maintain that its mere existence indicates that the intra-ocular pressure is raised above the normal intra-venous level, or the protrusion would not exist. Acting on this view, I have during the past year performed Iridectomy in four cases with hernial or cystoid cicatrices in which the pressure on palpation could not be said to be raised. The cases comprised two post-operative cicatrices—one after Glaucoma Iridectomy, and one after Extraction—and two cases of perforated corneal ulcer. In all four cases my iridectomy incision was made away from the cystoid cicatrix. As a result all the eyes have very much benefited, and the former cystoid cicatrices have flattened down, and appear now consolidated, showing that the internal pressure has been reduced to the normal intra-venous level, as otherwise the cicatrices would still be bulging out as before the Iridectomy.

SUMMARY OF CONTRADICTIONS TO FILTERING CICATRICES.

1. The intra-ocular pressure is derived from the circulation, and is not a product of intra-ocular volume.
2. The aqueous and intra-ocular venous pressures stand and vary together, but this parity would be destroyed if the aqueous filtered away.
3. The aqueous outflow is a resorption by veins, and not a filtrate, and therefore Glaucoma can only be prevented by increasing, as by Iridectomy, the area of contact between aqueous and veins.
4. The corneal endothelium always relines the inner margins of all incisions into the anterior chamber, and so effectively precludes filtration.*
5. Cicatrization of corneal wounds cannot be prevented by merely making the wound bigger by sclerectomy.†
6. The surface "œdema" consists of a hyperplasia of the transparent subconjunctival tissue which has supplied the cellular downgrowth which fills any gap, however made, in the scleral tissue.‡
7. A hernial or cystoid cicatrix indicates that the intra-ocular pressure stands above the normal intra-venous level.

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* See *The Ophthalmoscope*, December, 1907. † *Ibid.* ‡ *Ibid.*

PART V

A SURVEY OF THE SIGNIFICANCE OF EQUILIBRIUM BETWEEN INTRA-OCULAR AND INTRA-CRANIAL PRESSURE

In the Normal Eye (Fig. 58) the intra-ocular pressure is vascular in origin and nature, and varies simultaneously with the intra-cranial pressure, so that the same pressure exists on one side of the Lamina Cribrosa as on the other. The nerve and vascular elements connected with the visual function are thus subjected to a similar hydrostatic pressure throughout their whole course in eye and brain. This physiological association is of the utmost significance and value in assisting in the elucidation of a number of pathological manifestations in the eye whose causation is still obscure.

Glaucoma.

In Glaucoma (Fig. 59) the normal balance of pressure existing between the two sides of the Lamina Cribrosa is destroyed, as the intra-ocular pressure is raised above the intra-cranial, and therefore the weak Lamina Cribrosa recedes before even the slightest difference in pressure on its two sides, provided such is long enough continued.

Buphthalmos.

Buphthalmos (Fig. 60) is the infantile form of Glaucoma, in which through some anomaly, congenital or otherwise, the Cribriform Ligament becomes occluded early in life.

The same sequence of events leads to the intra-ocular pressure being raised in Buphthalmos as in the adult eye under similar circumstances, but, as the infantile corneosclera is less resisting than the adult globe, it yields before the raised pressure, and the dimensions of the eyeball are

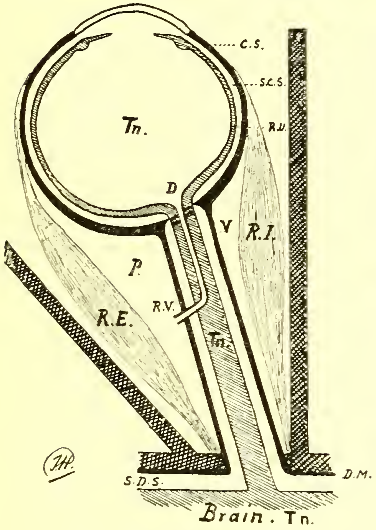

Fig. 58.—Normal Eye.

The tension within the globe and optic nerve sheath and brain all stand at the same level. The Lamina Cribrosa (D) is not required to support any pressure. The posterior pole of the Eye (P.) lies unsupported in the cone formed by the extra-ocular muscles, of which the Rectus Externus (R.E.) and Rectus Internus (R.I.) are here shown.

The Corneo-Scleral Envelope (C.S.), Supra-Choroidal Space (S.C.S.), Retina and Uvea (R.V.).

The central vessels of Retina (R.V.). The Vaginal Sheath of the Optic Nerve (V.), continuous with Dura Mater (D.M.), and opening into the Sub-Dural Space (S.D.S.).

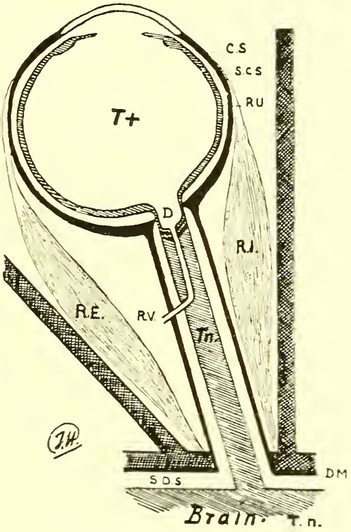

Fig. 59.—Glaucoma.

The intra-ocular pressure is raised, while the intra-cranial is normal; hence follows in time retrocession of the Lamina Cribrosa (D). The Retinal Vessels (R.V.), as they pass out of the globe into the Optic Nerve, are thus suddenly released from the raised intra-ocular pressure, resulting in manifestations of reaction being set up in the Optic Nerve. The rigidity of the adult Corneo-Sclera prevents a general yielding and enlargement of the globe.

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in consequence increased in every direction. The Buphthalmic globe may, in fact, be regarded as a generalized ectasia of the eyeball in which the Lamina Cribrosa recedes, as the intra-ocular pressure is maintained above the intracranial.

The term "Hydrothalmos," also applied to this condition, is a very suitable one, as it correlates the effect of increased pressure on the walls of the juvenile eye with the analogous results in the infantile cranium culminating in Hydrocephalus.

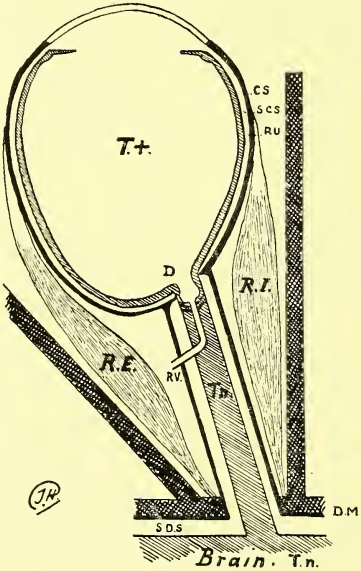

Fig. 60.—Buphthalmos.

The intra-ocular pressure of the infantile eye is raised above the intracranial; hence cupping of the Disc (D). The raised pressure within the infantile eye causes a general yielding of the Corneo-Sclera, and enlargement of the globe in all dimensions.

In the adult neither the coverings of the eye or brain readily give before increased internal pressure, but in the early months or years of life both eyeball and cranium readily yield before such pressure—a fact which should be added to the points of resemblance already enumerated as existing between the eye and brain.

Myopia.

The Myopic Eye (Fig. 61) owes its refractive state to an axial increase of the globe produced by an ectasia limited and circumscribed to the posterior pole. Many theories have been brought forward to account for the condition of retrocession of the posterior pole in Myopia, all being more or less based on the supposed action of the intra- or extra-ocular muscles in bringing about an increased intra-

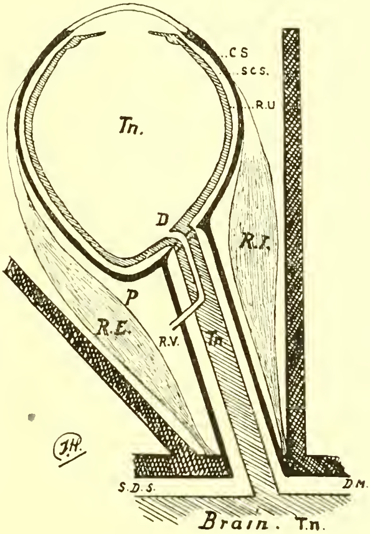

Fig. 61.—Myopia.

The tension in eye and brain are the same, and therefore Lamina Cribrosa remains *in situ*, and there is no cupping of the Disc. The greater axial increase of the globe after the fifth year is behind the equator, as by then the Cornea and Anterior relationships are fixed. In Myopia the unsupported Posterior Pole (P.) of the still growing Sclera yields too readily before the normal intra-ocular tension, thus producing an ectasia limited to the Posterior Pole.

ocular pressure. All such hypotheses must, however, be negatived by the one fact that the intra-ocular and intra-cranial pressures stand at the same level. Any increase in intra-ocular pressure in the myopic eye, however such may be hypothecated, would result in a dissociation between the intra-ocular and intra-cranial pressures, so that along with the yielding of the posterior pole there would also result a marked retrocession of the Lamina Cribrosa.

Cupping of the Disc is bound to follow any increase, however slight, of intra-ocular pressure, because the Lamina Cribrosa is anatomically far and away the weakest spot in the walls of the eye, and would certainly give way along with, if not before, the posterior pole of the Sclera. Further, however, the theoretical limitation of the effect of an hypothetical pressure to the posterior pole of the eye only is a physical impossibility, for, as already demonstrated, an increased hydrostatic pressure in the Vitreous is always immediately transmitted through the lenticulo-zonular diaphragm to the Aqueous.

Under all physiological and experimental conditions the Vitreous and Aqueous pressures always move together, and vary with intra-ocular venous pressure, so that it is impossible for the Aqueous ever to be retained in the Anterior Chamber at a higher level than that of the intra-venous pressure. The compression hypotheses of Myopia are altogether untenable, because compensation would always follow any intra- or extra-ocular muscular pressure on the contents of the globe of the eye. If such could not take place, the result would be Glaucoma, with destruction of the balance between the intra-ocular and intra-cranial pressures, and consequent cupping of the Disc.

In the myopic eye the intra-ocular pressure can never have risen above the normal physiological limits, because there is no retrocession of the Lamina Cribrosa, and therefore the intra-ocular and intra-cranial pressures must still balance each other. As the Lamina Cribrosa in the myopic eye is unaffected, the axial increase cannot be the result of a raised pressure, but simply of the ordinary physiological pressure acting on an abnormally weak and yielding tunic. Why the posterior pole should give way is apparent when the conditions which determine the normal growth in length of the eyeball are appreciated. The eyeball reaches its full size about puberty, but long before this, about the fifth year, the Cornea has reached its full development, so that during the intervening years the axial growth of the eye takes place solely behind the equator by a retrocession of the posterior pole.

One can, in fact, regard the growing scleral tunic as slowly giving way before the normal intra-ocular pressure till the physiological dimensions of the eyeball are reached. This normal elongation of the eye is facilitated by the fact that the posterior pole projects quite unsupported into a hollow cone (Fig. 58, *P*) formed by the Recti Muscles as they diverge from their origin about the optic foramen. In this connection it is instructive to note that in the adult eye the two areas of the globe which lie unsupported have the thickest walls, for the Cornea anteriorly, and the Sclera in the region of the posterior pole, have both a thickness of about 1 millimetre.

In the fact that the normal elongation of the eye takes place at its posterior pole, and that the intra-ocular and intra-cranial pressures stand at the same level, and vary together with all changing circulatory conditions, we have all the essential factors requisite to account for the excessive increase in axial length of the myopic eye (Fig. 61).

In a child's eye, anything that will tend to increase the physiological intra-ocular pressure, or diminish the power of resistance of the tissues generally, and of the connective tissue of the growing and relatively soft Sclera in particular, will cause an excessive yielding of the unsupported posterior pole of the eye, and therefore increased axial length of the globe. Head-work increases the intra-cranial and likewise the intra-ocular pressure, while unsatisfactory hygienic conditions (using these words in their widest sense) diminish the connective-tissue resistance of the scleral envelope. These are just the two most important factors at work in the production of "School" Myopia.

In Myopia the intra-ocular pressure is never pathologically raised, because the Lamina Cribrosa always remains in position, showing that the pressure in the eye balances that in the brain. The normal growth in axial length of an eye is behind the equator, and therefore if the Sclera, owing to some inherent weakness, gives way before the physiological intra-ocular pressure, such a globe will not only reach the normal physiological limits of size long before it is due, but it will rapidly overstep the bounds, and the optical and pathological results become manifest in Myopia. The normal intra-ocular pressure is more than the growing and weakened scleral tissue at the posterior pole can stand, and therefore it gives way in this locality.

The difference between the localized ectasia in Myopia and the generalized one in Buphthalmos is that the one results from the normal intra-ocular pressure acting only on a weakened and unsupported area of growing scleral tissue, the cornea being already fully developed; while the other follows from an increased intra-ocular pressure influencing at an early age the whole of the still growing tissue of the corneo-scleral envelope. The effect on the Disc in the former case is *nil;* in the latter, marked retrocession naturally follows.

Restoration of balance between the yielding tissue and the intra-ocular pressure results in the Myopia becoming stationary and fixed in amount. In other cases, however, the ectasia may go on increasing through all degrees and shades till the worse types are reached, in which all the deleterious effects of excessive stretching of the intra-ocular tissues are encountered.

The deformity of the myopic eye is the result of a weakened power of resistance of its supporting tissue to the ordinary demands made on it in the exercise of its physiological functions. Myopia thus stands in exactly the same category as other bodily deformities which arise from weakness in the supporting framework, as, for instance, spinal curvature or flat-foot.

In *Pes Planus* the arch does not give way before a pathological pressure, but through inability of the ligaments of the foot to support the ordinary body weight. The arch is not strong enough for its ordinary duties, and so gives way ; in other words, the deformity does not arise from any pathological increase of stress, but from a diminished power of resistance to strain. And exactly the same principles apply to the myopic globe in its yielding before the ordinary intra-ocular pressure.

One can carry the comparison between flat-foot and myopia much farther, however, for both conditions show the same age incidence as well as the same etiological unity throughout all their clinical manifestations. One meets with every stage and variety of flat-foot, from a condition amounting to little more than a mere tendency to the condition, to the extreme form in which the bones of the foot are profoundly altered in shape and position. And so, similarly, in myopia there exists the same unity between the "simple" type and its most malignant form. The same factors are responsible for the higher grades of myopia as for the lower ones, in which the power of resistance to strain is more marked. It is merely a question of degree, and not a difference in the nature of the causative agents.

Congenital causes and hereditary influences are considered by many authorities as playing a very important part in determining Myopia; and so they may, but to my mind to no greater extent than in producing flat-foot—to that extent only, and no more. In both conditions we are dealing with an exciting cause or stress which, although well within physiological limits, influences abnormally weak, "flabby" supporting tissues, which are unable to withstand such stress, and so give way. Weakness in supporting tissues arises from a multitudinous variety of general causes, of which, after all, heredity is only one among many competing influences.

Choked Disc.

The Choked Disc associated with increased intra-cranial pressure illustrates another type of the pathological results following dissociation between the intra-ocular and intra-cranial pressures. The manifestations so produced are, however, the very converse of those met with in Glaucoma, for the intra-ocular pressure remains normal, while the intra-cranial is raised.

The defects in the several theories advanced to account for the condition of Choked Disc must be apparent when we realize the physiological unity which exists in both eye and brain between their contents pressure and their intra-venous pressure. The vascular nature of the pressure within these two closed spheres makes it, in fact, quite unnecessary to consider any mechanical cause for the changes at the disc other than dissociation between the two pressures, and the effect such dissociation must have on the circulation as it passes from one level of pressure to another.

When intra-cranial pressure is raised, the intra-venous pressure throughout all the cerebral veins mounts to the same level, and the pressure within the optic nerve portion of the central retinal veins is no exception (Fig. 62).

Now, before the retinal circulation can continue through that part of the vessel enclosed by the vaginal sheath, the intra-venous pressure in the retina must rise to the same height as the pathological intra-cranial pressure. Hence arises the venous congestion at the Disc, which is directly proportional in amount to the rise in intra-cranial pressure.

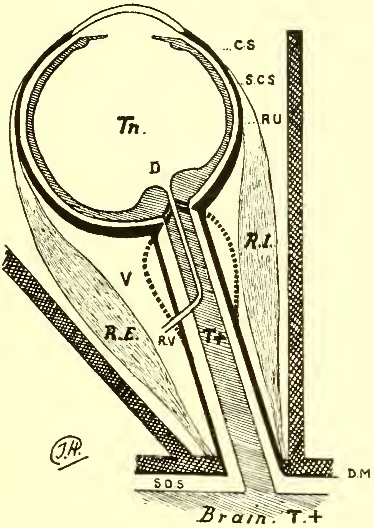

FIG. 62.—CHOKED DISC.

The intra-cranial pressure is raised, while the intra-ocular pressure is normal. To allow the retinal circulation to continue, the venous pressure within the retinal veins must rise to the same level as that within the cranium, and hence the venous congestion. The Disc (D) becomes œdematous, as fluids always tend to find their own level, and therefore percolate into its tissues. The Lamina Cribrosa will yield in time before the raised pressure behind it, while the Vaginal Sheath (V) of the Optic Nerve will become ectatic in its least supported area, which is just posterior to its attachment to the globe, as here it lies in the cone formed by the extra-ocular muscles.

The œdema and tumefaction of the nerve-head represent the degree and acuteness of the difference in the hydrostatic level between the two sides of the Lamina Cribrosa; in other words, the swelling of the Disc is the result of fluids tending to find their own level. The eye, from its lower hydrostatic level, acts in relation to the nerve-head and its circulation in the same way as an exhausted cupping-glass does when applied to the surface of the skin. The intra-venous pressure of the retinal veins comes thus to stand at a higher level than the intra-ocular pressure, and therefore exudation from those vessels follows, accompanied by extravasations in consequence of their unsupported walls giving way under their increased internal pressure.

The greater the intra-cranial pressure, the greater will be the cerebral intra-venous pressure, and therefore the more marked will be the manifestations at the Disc resulting from the disturbed parity of pressure normally existing between the eye and brain.

Besides these what we may call purely mechanical factors in the production of Choked Disc, we must not forget the fact that we are dealing with living tissues, and that the reaction of the delicate nerve elements to the altered conditions of their cellular life produced by the disturbed balance of pressure and changed circulatory conditions must be taken into account. It is this vital reaction, to what at the outset is essentially a mechanical process, which finds final expression in the loss of vision and an atrophic Disc.

The arching forward of the Lamina Cribrosa which accompanies the condition of Choked Disc is the converse of the retrocession which takes place in Glaucoma, both being the direct result of a difference in hydrostatic pressure on the two sides of the membrane.

As in Glaucoma, the alteration of the position of the Lamina Cribrosa in Choked Disc takes time to develop, and therefore it is a more prominent pathological feature in chronic than in acute cases.

The distension of the Vaginal Sheath, like the bowing forward of the Lamina Cribrosa, is another and late secondary manifestation of the effect of the raised intra-cranial pressure resulting in a yielding of the nerve sheath before the excessive pressure. The Hydrops Vaginæ, in short, corresponds to an ectasia of the least supported part of the nerve sheath, which is that part immediately behind the optic nerve entrance, as here the sheath lies quite unsupported in the cone formed by the extra-ocular muscles.

Leonard Hill* has shown that the brain substance behaves as a viscous mass, and that therefore under conditions of increased pressure it may manifest a considerable amount of pressure discontinuity. This dissociation of pressure within the cranium is brought about and maintained by the tense structures of Falx Cerebri and Tentorium Cerebelli, which firm unyielding formations are able to support a pressure in one hemisphere higher than that in the other.

This experimental fact, when coupled with the view I have just put forward respecting the nature and causation of Choked Disc, is of great value in supplying an explanation for the diagnostic significance of the ipso-laterality of Choked Disc as maintained on clinical grounds by Sir Victor Horsley.†

Normally the intra-ocular and intra-cranial pressures are the same, but pathologically the pressure in one hemisphere of the brain, and therefore of the ipso-lateral optic nerve, may rise above that on the other side. Under such conditions it is on the ipso-lateral side that the manifestations of papillary œdema will appear first, and present the most marked changes.

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* *Loc. cit.*

† Sir Victor Horsley, *Brit. Med. Journ.*, 1906 and 1910; "Trans. Ophth. Soc. United Kingdom," vol. xxviii.

GLAUCOMA: A SURVEY

Physiology.—In the rigid *cranium* the pressure of the fixed intra-cranial volume stands and varies directly with the cerebral venous pressure (p. 84).

Simultaneous measurement shows that

Intra-ocular pressure = Intra-cranial pressure (p. 85).

Therefore the corneo-sclera acts as an unyielding case with a constant volume, and the intra-ocular pressure is the same as the intra-ocular venous pressure (p. 89).

Hydrostatics.—Fluids always tend to the lowest hydrostatic level.

Therefore the fluids in eyeball and cranium lie at intra-venous pressure, because such is the lowest circulatory pressure in these closed spheres (p. 95).

Model of Normal Eye.—Total volume constant. Elastic circulatory system. Diffusion takes place between contained fluids and return (venous) circulation.

Therefore contents-pressure = return (venous) circulatory pressure of an *elastic* system of tubes (p. 98).

"*Glaucoma*" *Model.*—Total volume fixed. Absence of diffusion between contained fluids and return (venous) circulation.

Therefore the fluid and incompressible contents act as a rigid volume, converting the elastic circulatory system into a rigid one (p. 130).

The outflow pressure of a rigid system is always higher than that of a similar elastic system of tubes.

Therefore the contents-pressure is raised, as the lowest circulatory pressure is that of a *rigid*, and not an elastic, circulatory system (p. 131).

Glaucoma.—Sclerosis of the Cribriform Ligament hinders diffusion of aqueous into Schlemm's Canal.

Therefore the intra-ocular contents (aqueous and vitreous) act as a rigid mass, which forces the elastic circulation into rigid lines, and, in doing so, alters the relative volume of blood in arteries, capillaries, and veins (p. 135).

In the eye hindrance to diffusion is not absolute, as in the "glaucoma" model, but relative.

Therefore, as the total vascular volume in the uvea becomes altered, the aqueous volume is displaced and the anterior chamber becomes shallow (p. 142).

Iridectomy.—Wounds of the healthy Iris Stroma never cicatrize.

Therefore a Glaucoma Iridectomy, by facilitating the direct contact of aqueous with iris veins, prevents the intra-ocular fluids from acting as a rigid volume (p. 183).

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